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### ANÆSTHETICS OVER THREE DECADES: A REMINISCENCE.<sup>1</sup>

By HARRY J. DALY,  
Sydney.

IN opening this Section of Anæsthesia at this the Ninth Session of the Australasian Medical Congress, I wish to express my pleasure in being selected as president of the Section by the Federal Council of the British Medical Association.

At Sydney in September, 1929, the first meeting of the first Section of Anæsthetics was held under the presidency of my old friend Dr. Gilbert Brown, who was last year honoured by Her Majesty for his many contributions to advances in anæsthetics in Australia. That was a most successful meeting and was really the factor in binding together those interested in anæsthetics from all the States.

The address which I shall deliver will be in the nature of a reminiscence, and as such I trust will appeal for the most part to a younger generation.

It is my purpose to speak of some changes that have taken place in the means and methods of general anæ-

thesia from about 1920 to 1950; for my active participation and interest in this branch of surgery has covered that period. The progress made over these years has been so great that it must be considered the golden age of this specialty. Because blood transfusion is part of the activities of the anæsthetist, I think some observations on its development may not be out of place.

Surgery and anæsthesia have marched forward over these years, each quickening its pace to challenge the other for the lead, but surgery could never move forward till anæsthetics prepared the way.

The scene before the 1920's must be viewed to appreciate the revolutionary changes never anticipated by earlier workers in this field. The art of anæsthesia was practised, but the science had to await discoveries in chemistry, physics and pharmacology to establish fundamentals.

Let us consider what was happening in Sydney shortly before and during World War I. Ether was the common anæsthetic given through mask from bottle, a method which the young, modern, well-trained anæsthetist might consider a cloak and dagger attack. However, it was, and moreover probably still is, the safest agent and method for obtaining satisfactory anæsthesia and relaxation, over long periods if necessary, with a minimum of apparatus—a gauze-covered mask, a bottle of ether and an artificial airway. The senior student was taught this simple method, which was sound and enabled him the more easily to appreciate and follow the signs of anæsthesia.

<sup>1</sup> President's address to the Section of Anæsthesia, Ninth Session of the Australasian Medical Congress (British Medical Association), August, 1955.

Another method of ether anaesthesia used for some years prior to, during and shortly after World War I was the old Clover's inhaler. This I never see amongst the discarded anaesthetic junk without a shudder, recollecting many a short plethoric man bluey puffing his jerky breaths back and forth into a half-empty bag through the ether reservoir, dying for breath of air, yet living. In use, one breath in four or five was allowed to escape. The next was caught to refill the bag. This method was commonly used for major abdominal surgery. A surgeon removing the gall-bladder accepted the ebb and flow of the abdominal viscera with complacency. Little thought was given to accumulation of carbon dioxide. Later models of the Clover apparatus had a side inlet to admit oxygen.

Before the 1920's chloroform was often used for operations about the face and neck. The patient was first anaesthetized with open drop chloroform and a tracheotomy was performed. A gauze-covered funnel was then connected up to a tracheotomy tube by a three-foot length of rubber tubing. Chloroform was methodically dropped on the gauze to maintain anaesthesia. Such was a favourite method for Sir Herbert Maitland to use in his many extensive dissections of malignant glands of the neck.

Dr. E. J. Jenkins, a gentle-mannered physician on Sydney Hospital honorary staff who gave anaesthetics for Sir Alexander MacCormick, understood chloroform and "open ether" anaesthesia. He passed on only a few years ago, aged eighty-odd years. He used to dress near me at the Royal Sydney Golf Club. He told me that giving anaesthetics ruined all other forms of medical practice and advised me to discard it.

Early in the 1920's chloroform was fast disappearing. It was mainly used for induction prior to "open ether", occasionally in a mixture with ether or as a non-inflammable anaesthetic when some form of cautery was to be used. Many of the older surgeons bewailed the withdrawal of chloroform from the anaesthetic stage, missing the pale face and the bloodless field.

Ethyl chloride—"the gin-clear stuff in the small squirter-top bottle"—was used mainly for short surgical operations, local freezing anaesthesia or for induction prior to "open ether". We knew its sinister reputation—its high volatility might lead to early cardiac standstill. Instinctively we avoided its use in the debilitated or so-called vagotonic patient and the pale, fat child. We could not promise any patients that they would recover from the brief anaesthetic of ethyl chloride without a bout of vomiting. Yet we would not discard it even now, for its place might not be readily filled.

Before 1920, Dr. Mark Lidwill, now in retirement, was lecturer in anaesthetics at the University of Sydney. He was tutor in anaesthetics at the Royal Prince Alfred Hospital in 1909, where he was also later an honorary physician. Keenly interested in anaesthetics, he fashioned and assembled an ether vaporizing unit out of brass tubing and small gas fittings, adding a tank to contain ether and a mixing chamber of simple pattern to alter the strength of the ether-air mixture as desired.

This simple apparatus is still being used. Later, after some improvements were added, this machine was manufactured and distributed by a surgical house in Sydney. Many of these vaporizing units, modified to low pressure techniques, will be found in constant use in both private and public hospitals. Lidwill introduced the intratracheal anaesthesia method of ether insufflation at Royal Prince Alfred Hospital, though he himself points out (Lidwill, 1929a) that Thomas Flaschl, one-time surgeon to Sydney Hospital, was the first to use the method in Sydney, if not in Australia.

In addition to being tutor in anaesthetics, Lidwill was an inquiring physician especially interested in diseases of the heart and the developments in electrocardiography. He read a fine paper on cardiac disease in relation to anaesthesia at the first meeting of the first Section on Anaesthetics of an Australasian Medical Congress held in Sydney in 1929 (Lidwill, 1929b). He showed a portable electric

apparatus for stimulating the heart into activity. This he designed with the help of Major E. H. Booth, of the Physics Department of the University of Sydney. After describing its successful use in a stillborn infant he suggested: "This method of cardiac revival is applicable to the following types of cases, (1) Cardiac failure during anaesthesia . . ." As far as I can ascertain he had designed an apparatus closely resembling the modern defibrillator as used in the treatment of cardiac arrest.

Dr. Mark Lidwill blazed a trail and we are proud to claim him as a pioneer anaesthetist in Sydney.

#### The Period 1920 to 1930.

With this background let us view the scene between 1920 and 1930. Let it first be said there were no specialists in anaesthetics, that is, specialists who devoted their full time to anaesthetics. There were anaesthetists on the honorary staffs of the larger hospitals who combined these appointments with their general practice. Anaesthetists were in these years culled from those practising medicine rather than surgery, whereas now the anaesthetist is definitely allied with the practice of surgery. In private practice surgeons commonly chose physicians or junior surgeons to give their anaesthetics. Some of these physicians and junior surgeons were making tempting money from anaesthetics and so they gave it up with reluctance. Indeed, a few were nearly fenced in by the specialty and, fearing this, beat a hasty retreat. During this period the general practitioner who held an appointment as honorary anaesthetist on the staff of some large hospital, by constant practice became skilled and interested in anaesthetics and was sought out by *confrères* when they needed an anaesthetist for minor operations in the home or major operations in the hospitals.

This general practitioner was largely self-taught, reading the few articles on anaesthetics published in journals as a means of acquiring the latest advances. There were few modern text-books on anaesthesia to refer to in the early 1920's, and journals devoted entirely to anaesthesia were only just appearing. Some general practitioners were discovering that the giving of anaesthetics interfered so much with their general practice that, like the junior surgeons and physicians, they were forced to choose between specializing in anaesthetics or giving up their general practice. Thus there emerged out of general practice men who determined to make anaesthetics their career. This was a serious decision, for it meant giving up a reality for a shadow.

The experience gained from general practice was of particular value to the specialist anaesthetist because it gave him a wide insight into general medicine in all its aspects.

#### Nitrous Oxide.

During the 1920's nitrous oxide and oxygen were used mainly by the dental surgeon, often advertising painless extractions. Few medical men knew how to use it. As far as I am aware, the only surgical gas and oxygen machine was a Heidbrink at Sydney Hospital, and I recollect it was shown there at a clinical meeting of the British Medical Association about 1920. It was said to be used for only the worst surgical risks. I asked to be informed when it was being used, but apparently no one was interested in it and I do not know what happened to it finally.

In 1929 a modern McKesson apparatus was brought to Sydney by Dr. Kenneth Smith, of Darlinghurst, and before he used it, he exchanged it with me for a new Lidwill ether apparatus. I then possessed a gas machine, an eagerness to learn, and a book of instructions to guide and stimulate me. But I certainly had some alarming episodes, let there be no mistake about that. I had not seen a patient "saturated" with nitrous oxide, or rather "suffocated" from oxygen deprivation—only read about it. I did not think events would move so rapidly through the convulsive phase called "jactitation" to the stage when the eyeballs would be centrally fixed, the pupils wildly dilated, the face ashy-black, and yet recovery could be possible



when the colour returned after a blast of oxygen into the lungs. Nitrous oxide was tricky stuff to give successfully, but it had desirable features. Non-inflammable, acting speedily and pleasantly, no other drug leaves the body so rapidly and so smoothly as nitrous oxide. The brief period of anaesthesia needed for simple extractions makes it of special value to the dental surgeon. If thought desirable, any gaseous anaesthetic can be added to it to enhance its anaesthetic action. What matter now, that of itself it fails to produce the soft skeletal muscle? Its usefulness is widened by better machines, improvement in technique of administration, and the modern relaxants. It is an anaesthetic holding its place.

#### Blood Transfusion.

The difficulties associated with blood transfusion were often so great as to limit its use only to those cases in which it was most urgently needed. The citrate method, as it was called, was coming into use, but a method of whole blood transfusion, as recommended by Dr. Holmes & Court (1923), was being used, mostly in Sydney Hospital.

In this method whole blood was given from a modified Kimpton-Brown tube coated with paraffin. The paraffining of the inside of these tubes and caring for their sterility was a task for only the most skilled operators. The method and technique of receiving the donor's blood into a beaker containing sodium citrate as an anticoagulant was perfected by Lewisohn in 1915. The patient then received the blood by rubber tubing through an intravenous cannula from a funnel-shaped flask. Some thought it an advantage to bubble oxygen through the citrated blood during injection.

Cross-matching of numerous relatives as a supply of donors was also a problem, and many hours were spent, usually by the pathologist, seeking suitable donors. Meanwhile, many patients truly died of blood loss. Nothing, of course, was known of the Rh factor, and serious reactions not infrequently followed inaccurate grouping and lack of scrupulous asepsis.

In the earlier years the anaesthetist was seldom expected to attend to the transfusion, which was undertaken usually by a pathologist or a physician.

Commonly saline and glucose solutions were given intravenously to replace fluid volume to tide patients over the blood loss, but it seldom did in the desperate cases.

On looking through the *Transactions of the Third Session of the Australasian Medical Congress, 1929*, I have found scarcely a reference to blood transfusion as a restorative measure to combat blood loss as a first line of treatment. In a paper dealing with post-partum hemorrhage one author mentions warmth, absolute rest aided by morphine, raising the foot of the bed, and as regards fluid replacement therapy: "Fluids. Frequent small amounts of fluid should be given by the mouth and saline solution by rectum, subpectorally or intravenously. A novel suggestion is to use the patient's blood, if it has been caught, giving it with the saline solution by rectum. It is stated all the constituents but the corpuscles are absorbed." (Cameron, 1929.)

Severe blood loss, if the patient survived it, was treated by prolonged rest in bed with treatment of the anemia.

Such, then, was the position as regards blood transfusion in the 1920's in civil practice. The latter-day anaesthetist, with the welcome resources of a splendid blood bank at his easy call, can scarcely credit such a situation.

#### Ethylene.

About 1926 a new gas, ethylene, was used in Sydney. The late Dr. W. Vickers, in the search for a less disturbing anaesthetic than ether for use in operations on patients with toxic goitre, began to try ethylene in an apparatus imported by the Commonwealth Oxygen Company, of Foy Street, Balmain. It was called the "Safety Gas-Oxygen Machine". Everyone interested in anaesthesia was eager to try out this gas and this machine. Experiences with it

were written up in American journals (Sise, 1925), and at the Australasian Medical Congress, Third Session, Section of Anaesthetics, 1929. Was it the answer to a prayer for an anaesthetic giving relaxation combined with a rapid recovery and free from unpleasant after-effects? True, consciousness was lost after a few breaths, but stabilization in anaesthesia took some ten minutes to achieve. Relaxation was never good, and although recovery was only a matter of minutes, yet the patient was somewhat dazed and the smell could be detected in the exhaled breath for some ten minutes or more. This gas is lighter than air, and if much was used and escaped into the air the mixture had a distinct smell like the old acetylene lamp. Many theatre personnel complained of it and of the headache which frequently followed.

The late Dr. Stewart McKay, a rather touchy surgeon apt to be easily put out of temper, noticed the unusual smell after I had coaxed him to allow me to try it on one of his patients, having expanded on its virtues and freedom from later effects. I saw his nostrils dilate and, looking over his half-spectacles, he roared to inquire the source of this unusual and distinctive smell. Pointing at the machine, he asked: "Is the stink coming from that?" And when I had to admit that it was, he roared again that if he had been told so beforehand he would never have allowed it to be used in his theatre and, what was more, I was never to suggest using it again. Such were the trials of the anaesthetist.

Other surgeons were more reasonable under the same treatment, and so I was enabled to use ethylene over some six months or so, but then gave it up because it was not the answer to a prayer for an anaesthetic giving good relaxation combined with a rapid recovery and freedom from unpleasant after-effects. However, it was most useful for cases of short duration not requiring the relaxation that would be expected from ether.

No mention of ethylene was made at the Section of Anaesthetics of the Australasian Medical Congress, Fourth Session, at Hobart in 1934. It had lost its place mainly because of cost of machines and gas, poor relaxation, and unpleasant smell.

#### Rectal Anaesthetics.

Narcotics have been given as rectal injections for centuries. In the years between 1920 and 1930 drugs used commonly were paraldehyde or ether. The method of giving ether in oil to produce satisfactory analgesia in childbirth was introduced by Gwathmey in 1923 (Hunt, 1929). Such a method of producing basal narcosis or anaesthesia was tried by many anaesthetists in Sydney. Dr. Mark Lidwill said, in discussing rectal means of producing anaesthesia at the Congress meeting in 1929, that "the main objection was that the anaesthetic had to be begun on one day and completed two days later". Apparently the time factor ruled it out.

Paraldehyde as a rectal narcotic was good, but satisfactory basal narcosis was not always produced—part might be rejected, the dose too small or absorption poor. It became a popular premedication for children, following excellent results claimed at the Hospital for Sick Children, Great Ormond Street, London. If the child was docile and accepted the injection, the results were uniformly good, but poor if the child resisted and rejected even part of the drug.

After a favourable clinical trial by a group of anaesthetists chosen by the Medical Research Council and the Royal Society of Medicine, *The Lancet* of March 6, 1929, published a report on a new rectal narcotic of German origin—"Avertin".

An excellent account of the introduction and clinical use of "Avertin"—tribromethyl alcohol—was given by Dr. Kempson Maddox at meetings of the Section of Anaesthetics of congress in 1929 and 1934 (Maddox, 1929, 1934). He subsequently published a book on "Avertin". "Avertin" immediately became the rage. Patients asked for it. After a year or two it was somewhat abused and more than the basal degree of anaesthesia was attempted.

Patients occasionally failed to arouse for hours and at times were still asleep or lethargic after a week. Reports of post-operative pneumonia were published in *THE MEDICAL JOURNAL OF AUSTRALIA* (Darling, 1932). Except for cerebral surgery and neurosurgery it was seldom used after the mid-1930's. As a relatively safe and certain basal anaesthetic it has not been surpassed. Dr. Wesley Bourne, of Montreal, Canada, an authority on "Avertin" and its clinical use, recommended it as the best basal anaesthetic for children.

#### The Period 1930 to 1940.

##### Intravenous Anaesthetics.

As we enter the 1930-1940 decade a new chapter unfolds. The intravenous use of chloral hydrate by Pierre-Cyprien Oré in animal experiments led him to employ it as an anaesthetic in man. He was pleased with his results and published them in 1875 (Oré, 1875). The anaesthetic dose proved to be close to the lethal dose, but the long search had begun. It led to the use of derivatives of barbituric acid, first synthesized by Emil Fischer in 1902 (Fischer, 1903). "Pernoctan", "Nembutal" and "Sodium Amytal" were all tried in Sydney about 1931, after reports from abroad and supplies from local representatives of overseas manufacturers had been received. They were not satisfactory, some because of slow action, others because of delayed recovery. Of the short-acting barbiturates the first used in Sydney was "Evipan Sodium"—a startling innovation—effective, with rapid action and quick recovery into a pleasant dream state, soon to be followed by a feeling of well-being. I first saw it used by Dr. R. J. Silverton at the Royal North Shore Hospital of Sydney in 1933, when he gave it to patients requiring cystoscopy. We were all impressed. The total estimated dosage—averaging a gramme for an adult of ten stone—was injected in a matter of seconds, and this afforded some ten minutes' operating time. Drawing up of the legs, trembling, and slight degrees of "jactitation" with restlessness during the recovery period occurred at times, for the most part in men. It had a high measure of safety because it was rapidly destroyed in the body. Therefore it was most useful for operations of short duration and of a minor nature. It was also valuable to subdue the more resistant patients before the ethyl chloride—"open ether" sequence.

However, it was followed within a few years by the more desirable "Pentothal Sodium", which soon displaced it from the field both here and abroad. "Pentothal Sodium" was introduced only after prejudice and doubt had been allayed, for many surgeons, basing their objections on any intravenously injected anaesthetic drug, vetoed its use. They were won over sooner than expected, for patients themselves demanded it after glowing reports from all sides. It was much more potent than "Evipan", but displays of purposeless movements during anaesthesia were less frequently seen, especially if suitable premedication had been given and absorbed.

Long periods of anaesthesia with "Pentothal Sodium" were at first frowned on, but later it was found suitable for a wide range of operations, particularly those of a superficial character.

True enough, it had its limitations. The occasional case in which laryngospasm developed during the lighter stages of anaesthesia—the spasm often being caused by trickling of sour contents from stomach into the larynx—was startling and vexatious.

That thiopentone (B.P.) was by nature only an hypnotic was apt to be forgotten, and the anaesthetist was thus tempted to give overmuch in order to gain a degree of relaxation expected and afforded by ether. Respiratory and circulatory depression commonly followed such deep narcosis, especially if prolonged and accompanied by even slight suboxygenation.

Thiopentone has held its place at the front for many years, despite attempts to displace it. Truly, it has changed the outlook for both patient and anaesthetist alike.

##### Cyclopropane.

Cyclopropane, a gaseous anaesthetic investigated by Waters, of Madison, with his now well-known to-and-fro absorption canister, was introduced in Perth by L. A. Hayward and G. R. Troup in 1936. It was used the same year at Sydney Hospital in a Heidbrink closed-circuit absorption apparatus when Dr. M. P. Susman performed a total pneumonectomy for carcinoma. Fortified by contacts with American workers, and encouraged by Waters at Madison, Dr. Marshall and myself combined to present a paper on our experiences with this gas (Marshall and Daly, 1938).

Absorption technique with soda lime was necessary to conserve the gas, which cost about six shillings and sixpence a gallon, and three to five gallons were often taken from a cylinder for a single operation lasting for sixty to ninety minutes. It was all imported from America. We were soon aware of its potency and conscious of the wide range of explosibility when mixed with the commonly used high percentage of oxygen. The rather easily aroused parasympathetic activity appearing during anaesthesia made us wary and occasionally scared us, especially early laryngospasm and the cardiac manifestations, such as bradycardia, tachycardia and various arrhythmias. Complaints by surgeons of increased capillary oozing worried us because we knew not the cause or the prevention.

Induction was very rapid, usually smooth, and apparently pleasant enough. Maintenance was quiet, and though the signs of anaesthesia were less easy to recognize, generally the patient appeared comfortably asleep. Muscular relaxation following suitable premedication occasionally paralleled that gained from ether. Recovery was fast, but there was frequently a short bout of vomiting of yellowish gas-smelling bile, especially after short anaesthesia. We found it useful in quite a wide range of surgery—in thyrotoxicosis, diabetes, in surgery for children and the aged, in severe toxic states, and also during thoracic surgery because we had no relaxants and the surgeon got on without diathermy. It was often irksome to drag gas machines from hospital to hospital, but it was a good anaesthetic for the sick patient. However, as the range of "Pentothal" was extended, the simpler equipment needed, the pleasant induction and recovery, the elimination of explosion hazard and its combination with relaxants all combined to displace cyclopropane. High cost, greatly increased use of diathermy, less frequent use in teaching hospitals are other factors leading the anaesthetist to forget a satisfying adjuvant to nitrous oxide and oxygen anaesthesia.

##### Curare.

During the early years of the last war curare was introduced to anaesthesia. It was a drug from the jungle of sinister reputation and the idea of using it to relax skeletal muscles in the human was viewed with suspicion. We owe a debt to Richard Gill, the victim of a spastic disorder following an accident in the jungle, because he awakened the anaesthetist to the possible usefulness of curare in his book "White Water and Black Magic" (Gill, 1941). To Squibb and Company, of New York, who prepared "Intrococstrin", and to Griffith, of Montreal, Canada, who first applied it to anaesthesia (Griffith and Johnson, 1942), we are further indebted. Supplies were sent to me by Lieutenant-Commander L. H. Wright, of the United States Navy in 1943, but lay aside till 1945 because our surgeons would not accept it for trial in spite of many approaches. However, in 1945, Dr. Marshall and myself (Daly and Marshall, 1946) used it to supply the required relaxation needed to reduce the fracture and apply a splint to a patient suffering compound multiple fractures of the mandible. Soon afterwards surgeons began to accept curare as anaesthetists became better acquainted with its scope and limitations. That curare was not an anaesthetic, but inhibited somatic neuro-muscular transmission, had to be understood and realized. That it had a powerful quantitative action, but that we had a true antidote in prostigmin, likewise we learned.



The first lesson became evident when paralysis of respiratory muscles necessitated artificial support and control of ventilation if secondary circulatory failure was to be prevented.

It was evident that minimum anaesthesia was required such as could be supplied after suitable premedication by small doses of thiopentone and nitrous oxide; seldom was ether required, for it greatly increases the effects of curare and delays recovery.

The introduction of d-tubocurarine chloride, the active principle isolated by King in 1935, enabled a more reliable standardized preparation to be used than the original "Intocostrin" from Squibb and Company.

As a long-acting skeletal muscle relaxant with a satisfactory antidote in "Prostigmin" d-tubocurarine has not been displaced. Finally, I do not intend to speak of the advantages to the surgeon of induced hypotension or of hypothermia, each with its problems and anxieties for the anaesthetist, but will leave these to a future researcher, when their places in anaesthesia will be more clearly defined.

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#### PASSIVE PROTECTION AGAINST RUBELLA.

By HUGH WARD AND GRACE PARKER.

From the New South Wales Red Cross Blood Transfusion Service.

In 1941 Sir Norman Gregg published his observations that congenital cataract was associated with rubella contracted in the early months of pregnancy (Gregg, 1941). Other authors, notably Swan and his colleagues in South Australia, extended Gregg's original observation to the

association of rubella with other congenital defects (Swan, Tostevin and Black, 1945). These findings have been confirmed, although opinions differ about the frequency of congenital defects among children whose mothers suffered from rubella in early pregnancy.

Since the defects are serious and irreversible, it is obviously important to find a method of protecting women against the hazards of contracting rubella in the first three months of pregnancy. Although immunity is not absolute, one attack of rubella usually gives protection against a subsequent attack, and since rubella is a common childhood disease, the majority of adult women are immune. What can be done for the minority, who have no history of a previous attack? There is even a problem with women who do give such a history, because the clinical diagnosis of rubella is never certain and there is no laboratory test for immunity. In these circumstances, it would clearly be desirable to immunize women actively before marriage, if a rubella vaccine was available. Attempts are being made to produce this vaccine, but it is not an easy task.

Passive immunization by the injection of convalescent serum shortly after exposure to rubella would solve the problem, if this method could be shown to be effective in preventing rubella during the crucial early months of pregnancy. While it is the purpose of this paper to present evidence that passive immunization is effective in preventing rubella, other evidence, which will be discussed later, throws doubt on the value of convalescent serum in preventing the disease.

The Blood Transfusion Service of the New South Wales Division of the Australian Red Cross Society has made rubella convalescent serum available for the past four years to any doctor who applied for the serum, provided that (i) the patient was not more than sixteen weeks pregnant, (ii) the exposure was direct, and (iii) the serum could be injected not more than five days after direct exposure to rubella infection.

The dose of convalescent serum was fixed at 30 millilitres to be injected intravenously, and the serum was obtained from adult volunteers, who gave a donation of blood approximately three weeks after the disappearance of the rubella rash. Each blood donation was processed separately, because pooling would increase the risk of serum hepatitis. One donation provided six or seven doses of serum. The cooperation of the community in supplying this blood has been good, and only rarely has it been necessary to make a special appeal for volunteers.

Many hundreds of doses of convalescent serum were supplied to the medical profession; but, despite the demand and the apparent good results, doubts persisted as to the real value of the serum in preventing rubella. Accordingly, last year it was decided to make an attempt to obtain evidence which would stand up to statistical analysis for significance. The main problem in such an attempt was to find a sufficient number of comparable controls who had been directly exposed to rubella infection but who had not received the convalescent serum. If these could be found, it would be possible to compare the incidence of rubella in the treated pregnant women with the incidence of rubella in the untreated control group.

#### The Inquiry.

Two questionnaires were prepared and distributed.

The first, directed to the treated group, asked the following questions:

1. Patient's name.
2. Probable date of exposure of patient to rubella.
3. Date when injection of serum was given.
4. Did the patient develop rubella?
5. If the answer to question 4 is yes, date of first appearance of definite symptoms (rash, glands) of rubella.
6. Name of doctor who injected the serum.

This questionnaire was included in the package containing the convalescent serum, and the doctor who injected the serum was asked to fill in the questionnaire three weeks after the serum was given and return it to

the Blood Transfusion Service. The cooperation of the medical profession was excellent.

The second questionnaire, directed to the control group, asked the following questions:

1. Patient's name (or number).
2. Was the patient exposed\* to rubella at any time during her pregnancy?
3. Did the patient suffer from rubella at any time during her pregnancy?
4. If the patient did have rubella, at what stage in pregnancy did she contract the disease?
5. Was the patient injected with convalescent rubella serum?

(\* Exposed means that the patient (pregnant woman) was in the same room or vehicle as the individual suffering from rubella.)

This questionnaire was sent to private practitioners and to the ante-natal clinics at the large metropolitan hospitals. It was aimed at the women who were exposed to rubella in the later months of pregnancy and who had not received the serum. The last question was added to make sure that the women had not received serum. The medical officers were asked to fill in the questionnaires and return them to the Blood Transfusion Service. Again the cooperation of the clinics was excellent, although of course the great majority of women questioned had not been exposed to rubella and the completion of the questionnaires was tiresome.

This second questionnaire was also used in the Blood Transfusion Service itself; the questions (suitably modified) were asked of every female blood donor between the ages of eighteen and forty years, and the first question was restricted to exposure to rubella during the preceding twelve months.

#### The Results.

A summary of the answers to the two questionnaires is set out in Table I. In the first place, it will be seen from this table that approximately 90% of the control group did not contract the disease after direct exposure to rubella

TABLE I.

The Rubella Attack Rate in Passively Immunized and in Control Individuals

| Observation.                   | Passively Immunized Group: Pregnant Women. | Control Group.  |                        |        |
|--------------------------------|--|-----------------|------------------------|--------|
|                                |  | Pregnant Women. | Non-Pregnant Controls. | Total. |
| Number exposed to infection .. | 541  | 102             | 50                     | 152    |
| Rubella did not develop ..     | 536  | 91              | 44                     | 135    |
| Rubella did develop ..         | 5  | 11              | 6                      | 17     |
| Attack rate .. ..              | 0.9%                                       | 10.8%           | 12.0%                  | 11.2%  |

infection, and presumably the main factor in this resistance was a previous attack of the disease and the development of active immunity. Unfortunately it is not possible to determine at the time of exposure whether any particular patient belongs to the larger resistant group or the smaller susceptible group. The patient's history concerning a previous attack is unreliable, and in this investigation it was ignored both in the treated and in the control groups.

In the second place, it will be seen that approximately 1% of the subjects in the treated group subsequently contracted the disease. When this incidence of 1% in the treated group is compared with the 11% incidence in the control group, it needs no statistical analysis to prove that the injection of convalescent serum does provide a significant protection against rubella. Actually  $\chi^2$  is 40.64 for one degree of freedom, and the value of  $p < 0.001$ , which places the results beyond the realms of chance.

#### Discussion.

The results appear to show conclusively that convalescent serum gives a high degree of protection against rubella if

the serum is injected early in the incubation period. However, at least two objections could be raised against the material included in this inquiry.

The first was that the number of exposures in the treated group was too high, because the doctors who asked for the serum would naturally tend to err on the safe side and would inject the serum when in fact there was no effective exposure. In this connexion it should be pointed out that every request for the serum was reviewed by a medical officer in the Blood Transfusion Service and many requests were refused. Effective exposure was defined both in the treated and in the control groups as "being in the same room as the rubella patient". The serum was never supplied if the exposure was indirect—that is, through a third person.

The second objection was that the number of exposures in the control group was too low, because (i) non-pregnant women (the blood donors) would not be as conscious of exposure to rubella as pregnant women, and (ii) women in the later months of pregnancy would have ceased to worry about rubella and would not report an exposure. In this latter connexion, our own experience indicates that, although the knowledge of the dangers of rubella in pregnancy is widespread in the community, it is not generally appreciated that rubella is dangerous only in early pregnancy.

Again, if women at all stages of pregnancy were more conscious of exposure than non-pregnant women, one would have expected the attack rate among untreated pregnant women (because more exposures would be reported) to be lower than among non-pregnant women. In actual fact, Table I shows that there was no significant difference in the rubella attack rate in these two sub-groups of controls.

Anderson and McLorinan in 1953 reported a 1% incidence of rubella in over 800 exposed pregnant women injected with two or four millilitres of convalescent  $\gamma$ -globulin (Anderson and McLorinan, 1953), and the latter dose corresponds fairly closely with the 30 millilitres of whole convalescent serum used in our own work. While there is thus close agreement between Anderson and McLorinan's figures and our own in the incidence of rubella among pregnant women passively immunized, these figures in themselves mean nothing without controls, because 99% of adults might be immune to rubella without any passive immunization.

While we investigated the problem of controls in the manner described above, Anderson and McLorinan (1953) used another approach. In one experiment, volunteers who had no history of a previous attack of rubella were challenged with a spray of living rubella virus. Three days after the spraying, half the volunteers were injected with four millilitres of convalescent  $\gamma$ -globulin, the remainder serving as controls. In this experiment, the attack rate in the immunized subjects was actually higher than in the controls. However, the authors thought that in such an experiment the dose of virus might be considerably higher than in a natural infection, and this hypothesis is borne out by the overall attack rate of 50%, as contrasted with the 11% attack rate in our series of controls. It is fair to add that the volunteers were selected as likely to be susceptible, whereas our controls were unselected.

In another experiment, Anderson and McLorinan (1953) investigated the incidence of rubella in an epidemic in a Victorian boys' home. Half the boys (aged fifteen to eighteen years) received four millilitres of convalescent  $\gamma$ -globulin, the remainder serving as controls. Investigation of the ensuing rubella incidence showed a lower attack rate in the immunized boys than in the controls, but the difference was not statistically significant ( $p = 0.17$ ).

It would appear from these two experiments, first, that convalescent  $\gamma$ -globulin is not effective in preventing rubella in experimentally infected individuals, but the high attack rate of 50% suggests that it was perhaps not a fair test of the value of the  $\gamma$ -globulin in natural infection. Secondly, the result of the protection experiment in the boys' home epidemic, which was carried out most carefully, is suggestive, but does not present convincing evidence for the



protective value of the  $\gamma$ -globulin. However, the numbers were small—45 boys in each group—and the conclusions are based on contraction of the disease by three immunized boys as against seven control boys.

These Victorian experiments raise legitimate doubts about the prophylactic value of rubella convalescent  $\gamma$ -globulin, and we examined our own work—which leaves no doubt at all about the value of the convalescent serum—for flaws in the plan and conduct of the investigation. We can find no obvious fallacies.

In view of the findings reported in this paper, set out in Table I, and in spite of the doubtful results of the Victorian epidemic experiment, we must conclude that a dose of 30 millilitres of convalescent serum is effective in protecting pregnant women exposed to rubella infection. The only way in which we can reconcile our conclusive results with the equivocal result of the Victorian epidemic experiment is to suggest that the number of individuals at risk in the boys' home was too small for a conclusive result. It is to be hoped that an opportunity will occur for a repetition of the experiment on as large a scale as possible.

#### Summary.

1. Thirty millilitres of convalescent rubella serum injected intravenously early in the incubation period give significant protection against rubella.

2. On the other hand, previous experiments which have been reported (1953) throw some doubt on the value of passive immunization in preventing rubella.

3. This difference in the evaluation of passive protection is discussed.

#### Acknowledgements.

As the nominal authors of this paper, we regret that the New South Wales Red Cross Blood Transfusion Service can give no proper acknowledgement of the help received from many persons, help which has made this inquiry possible. In particular we are grateful to the convalescent volunteers who gave their blood, and to all those members of the medical profession who generously cooperated in completing and returning the *questionnaires*. We are much indebted to Dr. J. J. Graydon, of the Commonwealth Serum Laboratories, Melbourne, for his statistical criticism.

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### MR. CHARLES UNDERWOOD AND HIS ANTIDOTE, WITH SOME OBSERVATIONS ON SNAKE BITE IN TASMANIA.<sup>1</sup>

By W. E. L. H. CROWTHER, C.B.E., D.S.O., F.R.A.C.P.,  
Hobart.

The serpent subtlest beast of all the field.

Milton: "Paradise Lost."

SOME eighteen months ago a friend and patient, Mr. H. C. Tapping, handed me a small parcel, with the request that the contents should be presented in his name to the Royal Society of Tasmania. It contained a phial of "Underwood's Antidote for the Bites of Snakes and Other Venomous Reptiles", with a sheet of instructions for its use, wrapped in a piece of newspaper of the date 1870. Mr. Tapping explained that many years before it had

come into the possession of his father, at the time proprietor of the Freemasons' Hotel. This institution, recently rebuilt and one of the earliest in Hobart Town, still stands at the corner of Davey and Harrington Streets. It was for many years the property of the Tapping family.

About the middle of last century a surveyor and geologist, whom Mr. Tapping believes to have been a Mr. Gould, had a permanent room at the hotel, where he kept his effects, and to which he returned from periodical excursions to the interior of the island. This would have been Mr. Charles Gould, government geologist and son of John Gould the great ornithologist, who came to Van Diemen's Land in September, 1838, and spent several months collecting material relating to its birds. His wife, Elizabeth, was welcomed by Sir John and Lady Franklin and stayed at Government House, where her son, to be named Franklin, was born. With the completion of his investigations John Gould returned to the mainland, where his wife joined him in due course, and the united family left for England. There he prepared and published his monumental work "The Birds of Australia" in eight folio volumes, which is still the standard work on its subject. Until her untimely death in 1841, at the age of thirty-seven years, Elizabeth Gould was largely responsible for the drawing, botanical details and colouring of its incomparable plates (Figure 1). In the late 1850's their son Charles, all his life a great traveller, came to Tasmania, and for several years carried out geological surveys in its interior. The only hazard to an experienced bushman in our dense forest areas, apart from floods, accident or starvation, is snake bite; so when eventually Charles Gould left the Colony and certain of his material became the possession of Mr. Tapping, senior, it was not out of place to find with his case of drawing instruments the packet of antidote. The instruments are now in my possession, and the little bottle of Underwood's remedy, which was to have been the subject of a brief sketch, has instead become the centre of this lengthy narrative.

In the events that follow will be found references to diamond, black, brown, carpet and whip snakes. It is well recognized that the colouring of any variety of snake is influenced by its age, the season, and the environment in which it is found (Lord and Scott, 1924). Lord and Scott distinguish only three species for Tasmania—the white-lipped whip snake (*Hoplocephalus coronoides*), the superb or copperhead (*Hoplocephalus superba*) and the tiger snake, often misnamed black snake (*Notechis scutatus*). All are venomous, the tiger snake being the most deadly of the three. The two larger species can be differentiated only by the shape and position of the scales of the head. Thus all the snakes to be mentioned below, except the smaller whip snake, belong to the superb or tiger varieties.

#### Mr. Charles Underwood and his Antidote.

The actual remedy is about four drachms of a turbid fluid, brownish in colour and with a thick sediment, contained in a round glass bottle some two and a half inches in length and half an inch in diameter (Figure 2). The original sealing wax is over the head of the cork, and no attempt has been made to analyse its contents. The wrapper, measuring eleven by six inches, is undated, and bears the imprint "J. C. Hall, Printers, Advertiser Office, Hobart Town". The several testimonials printed on it are of dates from November 12, 1850, to January 20, 1859. The sheet is headed as follows:

#### Antidote

For the bites of snakes and other venomous reptiles.  
Price, ten shillings per bottle.

To be had of Mr. Millhouse, sole agent.

The undersigned, being in possession of a certain Antidote against the Bites of Snakes, and other Venomous Reptiles and Insects, tested in various experiments during a period of twenty years, is desirous of disposing of the same in small quantities for the benefit of the community at large. He is ready to submit his medicine to any test with rabbits, dogs, and other descriptions of animals, for the purpose of proving its efficacy in saving life before any competent person.

<sup>1</sup>Read at a meeting of the Section of History of Medicine, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

Directions for Use: Apply the liquid to the part bitten by a quill or any other method, and one application is quite sufficient for a perfect cure.

The testimonials which follow are of considerable interest and may be read with the aid of a glass (Figure II). It is of interest also that in each instance the snakes responsible are described as of the black or whip variety, no mention being made of the more deadly tiger. These letters to Mr. Underwood certainly indicate that his remedy was favourably known at this period throughout the country districts of Tasmania.

Of the man himself little seems to be known, and I have always understood that he was a Tasmanian and born in the Channel district or the Huon. The following piece of hearsay from the *Cornwall Chronicle* of November 29, 1859, was probably a "puff" to aid the sale of his wares:

Snake Antidote . . . another extraordinary cure has been effected by Underwood, upon the servant of Mr. Jessop of Muddy Plains, who was severely bitten by a snake some days since. There can be scarcely a doubt as to the efficacy of the antidote, from the numerous respectable vouchers which he possesses. The public are however too prone to neglect these discoveries. We understand Underwood acquired it in the Brazils, and it is well known how skilful the Indians of South America are in the cure of the bites of venomous reptiles.

Additional information as to Underwood's personality and methods may be found in the contributions of the late A. W. Hume to the *Tasmanian News* and *The Critic*. He was for many years their editor and an authority on local history. He was my old friend and patient. A most unusual character—sailor, prospector and journalist in turn, convivial and a good companion—he brought extraordinary qualities to the editorial chair. Roy Bridges and Carrel Clark, both outstanding historians, received their early training as journalists from him at the *Tasmanian News* office. Hume's last paper, *The Critic*, was published each Saturday evening, and included two columns concerned with Tasmanian history, both the work of the editor. They were headed respectively "Commentator" and "Notes by the Way", and were for the most part replies to correspondents. Requests for information on Underwood and his remedy were answered on various occasions between 1914 and 1924. One such reply is in the issue of March 3, 1924:

For some years the writer of this article possessed a bottle of Underwood's Mixture, but he never had occasion to use it. It was given to a resident of the Huon District but no one heard what use it was put to. It was a grayish mixture and one of its constituents was said to be the juice of fern roots. Vimpany (one time manager of the Sailors' Rest) often told me he used extract of fern roots largely. In colour Vimpany's mixture resembled that of Underwood.

A second, much more detailed, answer is of such interest that it should be given at length:

The writer has received a communication from a country subscriber (*Critic*, 31.5.1918.), asking if Underwood's antidote was ever proved efficacious in cases of snake bite. The reply is that an investigation was made by the medical authorities both in Hobart and Melbourne into the reliability of the antidote, and it was found to be useless as an antidote. The results of the experiments conducted at the General Hospital showed that more victims died with the antidote (applied by Mr. Underwood himself) than without it. For many years a Mr. Millhouse, who resided in Murray Street near the Wharf, vended the antidote, and it may be said that he honestly believed in its efficiency. In the year 1880 a person named Charles Garth, who resided at Port Cygnet, was bitten by a snake. Underwood's antidote was at hand and was applied, but it was useless as the man died. Underwood himself died of snakebite. (q.v.) . . .

Underwood was a man who seemed to have made a great study of snakes, which he juggled with in a most fearless way. In the year 1860 Underwood met his match in the snake charming line, when he paid a visit to Launceston. Here he encountered a Mr. Joseph Shires, who before Underwood's arrival, had been creating quite a sensation in the Northern city, with snakes of all descriptions brought in from the bush by disinterested parties. Shires, like Underwood, also claimed to have found an antidote, and there were thousands

of people in the North who were ready to announce that Shires possessed the secret of an effectual cure for snake bite. The meeting of Underwood and Shires was anything but a peaceful one. Underwood loudly denounced Shires as an impostor, and produced from his hat a black snake of about four feet in length, which he said if Shires would handle and allow to bite him would kill him in a very short space of time. Underwood then offered to give Shires ten pounds if he would let the snake bite him. He also said that he would go into the bush a short distance from the town, that they should look for snakes, and that he (Underwood) would catch the first one he saw with his mouth, and let it bite him, and that Shires should do the same, that Underwood would use his own antidote, and Shires that of his own invention. This was the test, he said, he was willing to undergo, and if Shires was willing they should set about it at once, whoever died from the effects of the snake bite the other to attend the funeral. Shires did not accept this challenge, and the result was that Underwood crowed loudly and Shires, who had able backers, came to the scratch and offered to meet Underwood in the Cornwall Assembly rooms, and give an exhibition of the value of their several banes and antidotes.

Underwood commenced operations by producing a large black snake from his hat, and Shires accepted the challenge to let it bite him. According to the chronicler of the day, Shires appeared to have much more confidence or reckless nerve than Underwood. The snake bit him on the little finger of the left hand. The antidote was then applied and he sustained no perceptible injury. He then invited Underwood to try one of his snakes, or rather, to let the snake try him. Underwood declined to comply at that time, Shires then allowed the snake he had offered to Underwood to bite himself on the upper lip, and again applied the antidote with the same result. The snakes were then tried on two kittens produced, but although the antidote was applied to one of them, it was not shown very clearly that either of them had been bitten, neither of them died.

After these experiments, Underwood left the room and was soon followed by Shires, who when passing Mr. Spearman's Inn, was invited by Underwood to step in. He complied, and in the presence of a considerable number of people, he twisted a large black snake around his neck, but that did not appear to offend it sufficiently to cause it to resent the familiarity. He said he would compel it to bite, and took a summary mode of coercing it. He bit the snake in the belly, about eight inches from its head, and when it curved its neck with pain and anger, gave Shires a severe bite under his right ear, and close to if not in, the Jugular Vein, the most dangerous spot in the whole body to have such a poisonous wound inflicted. The antidote consisting it was said of Tincture of Iodine and Liquid Ammonia, was applied to the wound which bled freely. In about ten minutes after he had received the bite, Shires appeared to become insensible, and seemed quite exhausted. He was driven to his lodgings, and was stupefied through the afternoon, from the effects of the bites. A few days after this episode another exhibition was given by Shires in the Cornwall Assembly Rooms, at which Underwood was present. Shires commenced operations with his own snakes, which did not appear to be very vicious specimens of reptiles. A black snake about four feet long was then produced by a man who said he had caught it on the Supply River, West Tamar. Shires invited Underwood to take the first bite of the stranger, but Underwood told Shires to go about his own business as he would have nothing to do with it. The new snake was a lively and dangerous looking one, and Shires, in taking hold of it, said if it killed him he alone would be responsible for his death. The snake quickly twisted itself around Shires' arm, but by careful manipulation he prevented it from biting him. He then carried it around the room and it was compelled to bite a dog. A number of persons at once clamoured to have the antidote administered, but the owner of the dog said that he was determined to see if the snake was venomous or not, and refused to have the antidote applied. The dog remained in the room for a considerable time, but the bite had produced no visible effect on the animal. At one period of the exhibition, the proceedings became disorderly, some of the audience evidently thinking the whole thing was a sham, and they had not got anything like their money's worth. In consequence of the irregular way in which the audience crowded around Shires it was impossible for



him to get fair play in making his experiments. Some of the furniture was broken, and once during the evening the platform, with snakes and antidotes, were all thrown down.

After this Underwood undertook a journey to Melbourne, but he failed to convince the medical men there that he had found the real Mackay. In his later years he drank heavily. What became of Shires history does not say.

#### Medical Research on the Snakes of Van Diemen's Land.

It must not be assumed that so important a matter as the scientific investigation of snakes and their venom had hitherto been neglected by the medical profession of the Colony. Two contributions to local scientific societies call for proper recognition. As early as May 25, 1843, James Agnew, M.D., Assistant Colonial Surgeon, presented a paper on this subject to the Tasmanian Society. He was

though perhaps in its details dry subject, that the following notes have been taken.

Dr. Agnew's observations are grouped under three sections—the bones and teeth of the lower jaw, those of the fangs and palate, and the poison gland and duct. He gives a vivid account of the fangs or poison teeth and of the replacement of this "terrific weapon" when broken off or decayed as a result of accident or injury, describing a large cavity, partly membranous and partly osseous, just behind the fang, where there are usually six rudimentary teeth in reserve. Of these, the most superficial are by far the more perfect and forward in development. As a result, when the fang decays, the reserve member advances and becomes fixed close to its side, and usually when the old tooth drops off its substitute is fully developed and ready for action. So, Agnew comments, "it will be at once evident that their [the fangs] extraction cannot, as was formerly supposed, render the snake permanently harmless". His remarks on the poison glands and their function are equally interesting and give evidence of most careful dissection and observation, as well as detailed anatomical knowledge. He describes also the three types of snakes used—the most common black snake, the rarer diamond snake of about the same size, and the whip snake, all of which he considers to have common characteristics, although the general dental arrangements of the last-mentioned were not minutely examined. His paper finishes in the following terms:

These are the only snakes I have ever seen on Tasman's Peninsula . . . they are all decidedly venomous. With the poison expressed from the gland I have succeeded in killing several small animals. On this subject, however, a complete series of experiments is much wanted, and many, not only interesting but important observations would certainly be the result. A wide and untrodden field of observation indeed still exists as to the entire natural history of these animals—the sizes of the different varieties—their appearance at different ages—the localities which they most affect—the presence or absence of the poison apparatus—the comparative intensity of the poisons of the different species, and how this may be modified by the season of the year and other circumstances—the exact time required for replacing an injured fang—their hibernation—mode of generation etc. all are important desiderata. Were observations on the subject however but generally conducted throughout the country by those who feel an interest in the subject, and authentic communications made from personal observations, a copious mass of important though isolated facts would very soon be collected, from which a satisfactory digest might easily be made, embodying almost every point of interest in relation to the natural history of the snakes of Tasmania.

This notable excursion into comparative anatomy is of great interest, and a worthy contribution to the scientific thought of so isolated a community. The same qualities are found in a second paper to the Medical Society of Victoria (Agnew, 1859). In this he describes how in 1848, when in charge of the surgical division of Her Majesty's General Hospital, he operated upon a man with caries of the shaft of the left tibia. Finding the disease to extend far more than had been expected, Agnew went on to remove every particle of the affected area, until in his own words "the denuded area was from six to seven inches in extent". The flaps were then laid down and water dressings applied with "but faint hope of having saved the limb". However, the patient made a slow recovery and was discharged in three months as cured. The paper reveals the careful appreciation of a sound surgeon of the factors for and against such surgery at this period. Dr. Agnew did much to advance science, art and literature in Tasmania, and was for a term its Premier. He was also largely concerned in the introduction of the live ova of salmon and trout to the antipodes. For these services he was created a Knight Commander of the Most Distinguished Order of Saint Michael and Saint George in 1895. Six years later he died at Hobart in his eighty-sixth year.

#### Some Observations of Dr. E. Svarbreck Hall on Snakes and their Venom.

Just a year after the foregoing article was published there appeared in the *Australian Medical Journal* a com-

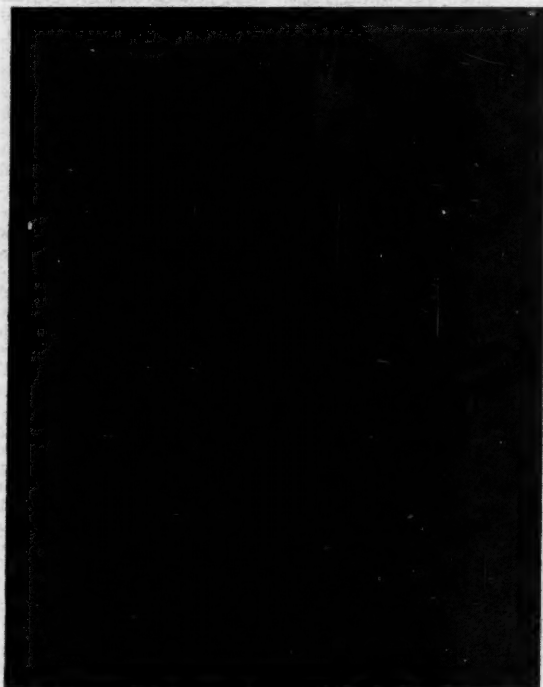


FIGURE 1.

"The Tasmanian Honey Eater", by Elizabeth Gould. (From a water-colour sketch in the possession of the writer.)

at the time posted to the establishment at Salt Water River, an out-station of Port Arthur, where coal of fair quality was mined by convicts for use on Tasman's Peninsula and export by small schooners to Hobart Town. The paper, entitled "Notes on the Teeth and Poison Apparatus of the Snakes of Tasman's Peninsula" (Agnew, 1846), opens as follows:

Having from time to time made a number of dissections in relation to the dental arrangements of the snakes of the Colony, and finding, at least in the books to which I have access, some anatomical points connected therewith, either misstated, omitted altogether, or not strictly applicable, I have been induced to throw a few notes on the subject, into a connected and continuous description of the entire dental apparatus. Important as the part is which these animals play in the zoology of Tasmania, their habits are still involved in a considerable degree of obscurity, and their nature is but little understood. I have even indeed heard their poisonous qualities made the subject of doubt, and it has been during an investigation of this interesting,

munication entitled "On Snake Bite" (Hall, 1859). Its author, an honorary corresponding member of the Medical Society of Victoria, had been for many years an assistant colonial surgeon stationed in various country districts of Van Diemen's Land and for a period superintendent of Her Majesty's Colonial Hospital. The late Dr. J. H. L. Cumpston (1923) was the first to recognize the extraordinary part that Dr. Hall played in preventive medicine, as it is now termed, in Tasmania. Indeed, he went so far as to refer to him as "the first Australian sanitarian". Hall wrote principally on the relationship between climate and disease, making Van Diemen's Land his laboratory. His papers appeared in *The Journal of Epidemiology*, the *Australian Medical Journal*, and the *Proceedings of the Royal Society of Tasmania*. He contributed as well to the local Press on matters concerning the health of the community. To silence detractors of his Catholic faith, Hall (1875) published at Hobart Town a most scholarly work, "Who Translated the Bible?", using in its compilation his own collection of early Bibles and other sacred writings. Hall died at Hobart on August 9, 1881, a month after the celebration of his golden wedding and forty-eight years of practice in Tasmania. The felicity of his literary expression will be appreciated by the extracts from his paper that follow. The brief title gives little indication of the scope and real scientific interest of the article. It opens with the description of a recent case of snake bite treated at Her Majesty's General Hospital, Hobart Town (the change of title seems to have followed its transfer from the Home Government to that of the Colony). The accident, its clinical course and the post-mortem findings are given in detail, after which Hall continues with a fascinating account of his experiences of the incidence and treatment of snake bite in Tasmania.

The victim, George Henwood, was a watchmaker by trade, who wandered over the country to repair clocks and watches. He also, in Hall's words, "proffered himself as a snake charmer, carrying snakes about his person for exhibition at races, in Inns, and other places; while at the same time he endeavoured to sell bottles of a fluid, which is here familiarly known as Underwoods Antidote for Snake bite". On the day of the accident Henwood was exhibiting a black snake and a diamond snake, when the former was teased and induced to bite a chicken.

Whilst this was going on the diamond snake glided away into a rose bush. Henwood seized the deserter by its tail when it turned round and bit him on the inside of the index finger of the right hand. Having sucked the wound, he cut the finger and taking a small bottle from his pocket Henwood rubbed some of its content on the finger where it was bleeding.

He also sent one of the company for some twine with which to bind his wrist, and some gin. He also sent for the chicken (already bitten by the black snake) and "applied the fangs of the diamond to its comb". The chicken died in two minutes, when Henwood desired the Inn keeper to destroy the snake, which he did. Poor Henwood then became ill and took to his bed, and his pain and general condition showing no improvement twenty-five hours after the bite, he sent for medical assistance from the city. He was found to have, *inter alia*, signs of tetanus about the mouth, and as treatment *Liquor Ammonii* was applied to the wound, a strong purge was ordered, to be followed by ammonia and ether, and for good measure brandy and water as well as broth. A night of pain and restlessness followed, and on the next day Henwood was removed in a chaise cart from Mr. Shadwick's inn to Her Majesty's General Hospital. Here he came under the care of Dr. Brock, who, finding the signs of tetanus to have lessened, ordered a mustard emetic, to be followed by *Liquor Arsenicalis*, after which turpentine and oil were to be rubbed all over the body. That done, Brock handed the unfortunate Henwood over to Dr. Eckford, who had some years before treated him for snake bite at the Impression Bay Station (Tasman's Peninsula). Eckford stated that on that occasion Henwood had been invalided for an injury to his left leg. At that time he professed to be a snake charmer, and "one day having a snake next his bosom, tripped and struck his chest

where the reptile was coiled, when it bit him over the Great Pectoral muscle near the shoulder". After the bitten portion had been widely excised by one of the officers and an ipecacuanha poultice applied, Henwood made a good recovery.

However, on the present occasion his condition steadily became worse, the rigidity of the mouth persisting although no tetanic spasms occurred. Drowsiness increased, with suppression of urine, restlessness and loss of power. Yet he remained conscious to the last, and being apprehensive that he would die, he informed Dr. Eckford that Underwood's antidote was an infusion of ipecacuanha and ammonia. Death occurred seventy-two hours after he had been bitten. An autopsy was carried out, and Dr. Eckford having scratched his finger at an early stage, it fell to Dr. Hall to complete the examination. The detailed report reveals Hall as a most competent anatomist, with a wide knowledge of pathology. He describes how, when the skin was reflected from the finger, "a puncture such as a pin might make was faintly observable penetrating the digital vein, but it required the aid of a magnifying glass to discern it satisfactorily; when the vein was slit open, the fact of its being pierced through was confirmed". A detailed description of the other organs followed, and stress was laid on "the fluidity of the blood and absence of fibrinous clots". "The nose of a cat was inoculated with blood from the veins of the bitten arm, but no injurious effects ensued." Hall also adds that Dr. Eckford did not suffer any evil consequences from the scratch he had sustained.

Henwood's case concluded, Hall passes on to review five instances of snake bite treated by Dr. J. Doughty of Oatlands, who had been its Colonial Surgeon for fourteen years. The persons concerned all recovered by the use of stimulants and application of ligatures, with excision of the wound and its excoriation with gunpowder. Here, too, may be mentioned a short memo on snake bite which appeared in *The Guardian* of January 22, 1851, under the initials "T.C.D.". It is very probably written by a medical man, possibly by Doughty, although the initials do not agree. The correspondent mentions that Mr. Underwood had been at Oatlands some weeks previously, exhibiting at a public inn. Of two fowls that he had caused to be bitten, one that received antidote died before morning, while the other without its help was alive and well. The author then briefly described two cases of his own; both patients had been bitten, and one of them died in spite of treatment. After an observation that the "secret" might be obtained from Underwood or any of the "snake doctors" for a small fee, and that he had known of such secrets being sold for a bottle of rum, "T.C.D." went on to predict that:

To an Electro Galvanist or Mesmerist, the honour of the discovery of the true antidote will probably fall. In future experiments, I shall try the Vaccine Lymph, with the view of counteracting the poisonous matter of the snake, and whether successful or not you shall know the result.

To return to Dr. Hall's paper: He next describes a series of three patients treated by himself, one of whom died. The first, a shepherd, was reported to have been chased and bitten by a large snake, and to be "swelled up, vomiting, fainting etc etc", although the wounded part had been cut out, sucked and burned with gunpowder. Unable to leave his duties, Hall sent "Ammonia, Aether and Liq Ammon Fort for external application and ordered spirituous stimulants". The shepherd, after having taken a few doses of the latter, began to rally, and in a few hours was quite well. The second instance occurred some years later, when a boy, aged twelve years, "who was in the habit of snareing mice between the niches of the rude slab floor in a shepherd's hut . . . thought he saw a mouse's head dart up and bite him in the second finger of the right hand. He rapidly became ill and not being treated died in eleven hours". Quite unsuspecting of the real nature of his injury, the shepherds subsequently took up the flooring, and found a diamond snake lying coiled up there, which they killed. "It was nearly five feet long with very large poison fangs, when I examined it", Hall stated, and continued:



I made a very minute autopsy of the boy. The extreme congestion of every organ existed as in Henwood's case, but in a much more aggravated form. The minute mapping of the vessels of the Pleuro Costalis exceeded anything I had ever before witnessed. No artificial injection that I ever saw in the dissecting room was nearly so beautiful. The poison fang had, in this boy's case, passed directly into the digital vein, but by a very large orifice. On the opposite side of the finger there were two minute wounds, from the prehensile teeth. There was a very considerable serous infiltration, both in the finger, forearm and upper arm. Some of the absorbent vessels were red and full, but the glands were unaffected.

snakes and enabled me to slay great numbers of them. In 1848, I made a collection for the Museum at Chatham. The reptiles were brought to me alive, that I might destroy them scientifically, and without injuring them as specimens. The men who caught them for me would pin them to the ground with forked and pointed sticks, and tie them to the stick. I put them to death with a poison as potent as their own, the oil of Tobacco. This I procured by moistening a sharply pointed splinter of pine with saliva, and then rubbing it on the inside of the stem of a well used tobacco pipe. The snake touched with this anywhere within the mouth, would speedily become convulsed and in a few minutes expire. Snakes of upwards of five feet long were so destroyed

*The under-mentioned Testimonial has been gratuitously offered me.*

**"Hoon River, Nov. 12, 1850.**

"Sir,—I have to bring under your notice for the benefit of Society, the fact that a man of the name of William Gibbs was bitten severely in the right leg by a large Black Snake, which he accidentally trod upon. The leg soon became greatly swollen, and in a short time he lost his senses, and remained in that state until Underwood's Antidote was applied, which was fully successful. After the bite was given, when shortly afterwards his senses, soon felt relief, gradually recovered, and is now in a perfect state of health.

**WILLIAM GIBBS.**

**"At Mr. William Wood's."**

**Testimonials.**

About three months ago, in the town of Gregson, M.G., Mr. George A. Ken... other persons, a Snake of the whip... the thumb, which I cured by the application of the Antidote. The same Snake bit a Dog... species in the ear. The Dog lived for four days and then died. The above gentlemen have allowed me use their names as reference.

**CHARLES UNDERWOOD.**

**January 1, 1851.**

**SHAKES.**—On Friday last, a man in the employ of Mr. Gordon, of North West Bay, was lifting a log, when a Snake bit him in the fleshy part of his arm, and hung on it. The men shook it off, and wished his mate to cut a piece of flesh out of his arm; but he being afraid of injuring some of the blood vessels made but a small incision. He then made the best of his way to a medical gentleman, who was not at home,—and hearing that Mrs. Fisher had some of Underwood's Antidote, he went to her, and some

**UNDERWOOD'S SNAKE ANTIDOTE.**  
To the Editor of the Advertiser.

DEAR SIR,—Between nine and ten o'clock on Sunday morning last, on the North West Bay Road, near Kingston, a little boy, aged about three years, walking barefooted, trod upon a large Black Snake, and was bitten on the sole of one foot. The child was immediately carried to the township, and taken into the cottage of Constable Riley, where Underwood's Antidote was freely applied to the wound, in presence of Mr. Chief District Constable Fitzsimmons, Mr. Keen of this place, myself and others. The child, until the application of the Antidote, appeared to be drowsy, and seemed to take no notice of the bite; but immediately on its application, he cried to have the stuff taken away, and suffered such acute pain, that it was thought he was going into convulsions, and medical aid was sent for. The pain, however, gradually ceased in the course of the next few minutes, and after the lapse of about half-an-hour, he seemed quite recovered, and has continued so ever since.

The boy (who is an intelligent little fellow) in answer to an enquiry of mine, pointed out—while suffering from the pain of the Antidote—the place where he was bitten, and said "the Snake did it"—a wound—which was a jagged one—also bore appearance of being such as would be caused by a large Snake.

Every reason to believe that the application of the Antidote saved the child's life, and, I think, that circumstances should be made public, through the medium of the value of the preparation may be by residents in the bush.

Other state, that some years ago, I was with success, to a finger of a man who had been bitten by a Centipede. On the finger had commenced swelling and was painful. The application of the Antidote caused the swelling to subside, and after the lapse of an hour, nothing more was felt of the effects of the bite.

I am, dear Sir, yours faithfully,  
**E. INNES.**

**Police Office, Kingston, Nov. 7, 1853.**

FIGURE II.  
The bottle of Underwood's antidote.

The next victim, having been bitten on the finger by a whip snake, took a tomahawk, and laying the bitten member on a log, chopped it off. Hall drove him in his gig to the hospital at Oatlands, where:

By great pains (he) succeeded in making a good stump to the finger, without putting the poor fellow to the torture of a second operation (secundum artem) after his own prompt and resolute bravery in operating.

Dr. Hall continues with an account of his own experiences with snakes, and the narrative must be given in his own words:

I have had favourable opportunities in many parts of the Island, of judging of the abundance of snakes. At the time when Lady Franklin so philanthropically gave a shilling each for every snake destroyed, I saw great numbers brought to country police offices for the reward. A fine retriever dog I brought out with me from England, became a most inveterate hunter of

... I have met with only four species of snakes in Tasmania, the diamond, the black, the green or whip and the carpet snake. The two first I have found equally abundant, wherever I have been. In the Westbury and Richmond districts, I saw the most whip snakes. Of the carpet snake, I only saw one specimen, brought to me alive, when I was in medical charge of the Invalid Station at Jerusalem. It was beautifully mottled and variegated in colour, and nearly five feet long. It corresponded with the description of the reptile of the same name in New South Wales. This specimen, however, could scarcely have been imported with any ship's cargo from the older colony, for the locality where it was captured is in the interior, thirty miles from Hobart Town or any part of the sea coast. The specimen was preserved for the Chatham Military Museum.

In warring unto death with these dangerous foes, I have had many narrow escapes. On one occasion, having failed in disabling a large opponent by the first

stroke of my gig whip, it attacked me so furiously that I was obliged to retreat from it, in a similar attitude, but much more hastily, than I should have done from the presence of royalty. In doing so I stumbled over a hillock, and fell, heels upward, facing my enemy. The snake at the moment was erect for a spring, and quite close enough to reach me, but seemed to be so utterly dumbfounded by my sudden change of position that it did not advance. I can scarcely attribute this to that courageous disdain, that scorns a lifeless foe; but by this delay I was enabled to regain my feet and succeeded in despatching the bewildered creature.

After an account of another incident, in which one of his children narrowly escaped from a large snake in the garden, Dr. Hall continues with reflections on the habits of these reptiles and their size:

The largest snake I ever saw measured was killed with a stockrider's whip, on the high road, by a gentleman riding with me. It was of the black species and seven and a half feet long. But I believe that once I saw one that measured by my eye, must have been nine feet long and thick in proportion. It was put up by the dog I have alluded to, in low scrubby unfrequented ground near a watercourse, but my horse was so restive that the snake escaped before I could aid my dog in the destruction of the monster. Four to five feet is a common length for the black and diamond snakes, eighteen to twenty-four inches for the whip.

Hall naturally was familiar with the work of Dr. Agnew, and discusses his conclusions as to the automatic injection of venom when the snake uses its fangs. He himself inclines to the theory that Tasmanian snakes "can inflict at their pleasure" an ordinary puncture wound or an empoisoned one. Towards the end of this most interesting paper there is a brief appreciation of an article by Dr. Roberts in *The Sydney Magazine of Science and Art* (November, 1857). Dr. Hall concludes with a summing up of Henwood's case, suggestions for the proper handling of snakes by such people as Underwood, and a strong plea for the further study of the effects of snake venom.

#### *The Later Years of the Century.*

This narrative so far has been concerned with the incidence and treatment of snake bite in Tasmania alone. It is very probable that each of the other Australian colonies had similar antidotes, if not an Underwood. Mention should certainly be made of two pamphlets published in Victoria (Berncastle, 1868, 1869). Both have as their subject the treatment and cure of Australian snake bites. The latter is a reprint of a paper read before the Victorian Medical Association on February 12, 1869. Its predecessor, for full measure, has in addition an article on the use and abuse of tobacco. This was read before the Royal Society of New South Wales on November 11, 1857, the president, Sir William Denison, in the chair. Yet another lecture by Berncastle on snake bites (not seen) appears in the *Transactions of The R.S. of Sydney* (sic), Volume 1863-4. In the earlier pamphlet, after some general remarks on snakes and their bites, is a summary, "Dr. Berncastle's directions for the successful treatment of all Australian snake bites". The eight directions are very briefly as follows: (i) The sucking of the wound and use of ligatures. (ii) Excision of the area of the bite. (iii) The administration of stimulants.

A pint of Brandy by the wineglassful at intervals of a quarter of an hour. A quart bottle of spirits will not often produce intoxication in an adult after a snake bite, but ensure the cure. In the absence of spirits the half pint bottle of my Antidote, a half ounce of Liq. Ammonia, the same of Tr. Lavand Co to seven ounces of Sp. Vini Rect, will be found equivalent to a bottle of Brandy.

- (iv) Rest. The patient must sit down all the time with plenty of fresh air, and his spirits must be kept up by cheerful encouragement of success by those around him.
- (v) The treatment of the wound by stopping hæmorrhage, and by the application of dressings and sticking plaster.
- (vi) The application of the actual cautery, such as a thin red-hot wire, to the bitten part in certain cases.
- (vii) Quickness in action, the golden rule in all cases.

Dr. Berncastle appears to have enjoyed writing, and among my papers is a pamphlet by him on "The Revolt of the Bengal Sepoys", published at Sydney in 1857 and presented by the author to Dr. William Bland. Berncastle describes himself on its title page as "author of travels in India, China, South Africa, etc. etc.". He kept his name well before the public also, and *The Sydney Morning Herald* of December 18, 1862, describes a box he has had arranged expressly for the personal use of people living in the country. In the compartments of the tin container designed for the purpose will be found a half pint of the spirit antidote, a knife, a pair of forceps, lint, ligatures and sticking plaster, with printed directions how to proceed without waiting for the doctor; quite an attractive advertisement for Christmas week, in fact.

In Tasmania the subject finds its way into the Press from time to time, as in *The Mercury* of January 29, 1881, when Underwood's henchman, the worthy Mr. Millhouse, replying to a correspondent who had condemned all snake cures, made the excellent rejoinder that any want of success in their employment was due to the remedy's not having been properly applied. Having supplied particulars of recovery following its use, Millhouse continued:

It is true that Underwood died from snake bite, but your correspondent does not tell you that the antidote was not properly applied. Let him read the inquest. But I am in a position to tell him that it was not properly applied, he being under the influence of Liquor at the time.

On February 19, 1881, again in *The Mercury*, L. Grey Thompson, M.B. of Launceston, writes in reply to one "Armidillo" and other correspondents. He supplies the information that Underwood died on December 29, 1881, after having been bitten by a carpet snake which had no fangs and was innocuous, his death being due to heat apoplexy. Some weeks previously he had had a similar fit. He was at the time aged over sixty years and was intemperate. Dr. Thompson then goes on to mention Lucroft's remedy (perhaps an antidote evolved in our sister colony), and an experiment in which a large Victorian tiger snake was used. He suggests the possibility that a relative immunity might be present when the subject was bitten for the second time, although Lucroft himself had suffered severely after such an experience. Thompson's own cat, bitten one day, survived with the application of that remedy, only to die the following day from a second bite of the snake described above. After mentioning the varieties of Tasmanian snakes, Thompson mentions the copperhead or large-scaled snake, stating that "it was one of the latter that bit the late Dr. Valentine". Later in life Dr. Grey Thompson became Health Officer for the City of Launceston, and the writer recalls warm memories of his personal qualities and hospitality, when working under his direction, in a temporary appointment to its Quarantine Station, during the small-pox emergency of 1913.

To return to the eighties: On October 27, 1884, *The Tasmanian News* reported a visit made to Green Ponds (now Kempton) by a party of gentlemen which included Dr. Agnew and Mr. Alexander Morton, curator of the Tasmanian Museum. Their purpose was to inquire into the efficacy of the antidote used by one Phillips, a local charcoal burner. After some delay due to a demand from "Old Charley" for some money prior to the tests, Phillips took a snake from a box and handled it freely. The serpent then turned and bit him savagely on the fifth finger of the right hand. The report states:

The old man treated the matter with the utmost sang froid and watched the blood that flowed freely from the puncture. He then coolly applied his antidote, and beyond a slight swelling of the hand no ill effects were felt from the bite, and the discoverer is as well as ever.

The snake was brought to Hobart and dissected by the Honourable Dr. Agnew, M.L.C., and the poison glands were found to be intact and not tampered with in any way. The account concludes with the statement that the test will be renewed "this afternoon" at the Cascades, with Mr. Morton and Dr. Agnew in attendance, and expresses the



# ANTIDOTE

FOR THE BITE OF SNAKES AND OTHER VENOMOUS REPTILES.

PRICE, TEN SHILLINGS PER BOTTLE.

TO BE HAD OF MR. MILLHOUSE, SOLE AGENT.

**THE UNDERWOOD.** being in possession of a certain Antidote against the Bites of Snakes, and other Venomous Reptiles and Insects, tested in various experiments during a period of twenty years, is desirous of disposing of the same in small quantities for the benefit of the community at large. He is ready to submit his medicines to any test with rabbits, dogs, and other descriptions of animals, for the purpose of proving its efficacy in saving life before any competent person.

**Directions for Use.**—Apply the liquid to the part bitten, by a quill or any other method, and one application is quite sufficient for a perfect cure.

The undermentioned Testimonial has been gratuitously offered me.

"Hoon River, Nov. 12, 1850.

"Sir,—I have in being under your notice for the benefit of Society, the fact that a man of the name of William Gibbs was bitten severely in the right leg by a large Black Snake, which he accidentally stood upon. The leg soon became greatly swollen, and in a short time he lost his senses, and remained in that state until Underwood's Antidote was applied, which was fully three hours after the bite was given, when shortly he came to his senses, soon felt relief, gradually recovered, and is now in a perfect state of health.

WILLIAM GIBBS.

"At Mr. William Woolley's, Bullock Point."

Testimonials.

About three months ago, in the presence of Mr. Gregson, M.D., Mr. George A. Kemp, and several other persons, a Snake of the whip-scorpion bit me in the thumb, which I cured by the application of my antidote. The same Snake bit a Dog of the cur species in the ear. The Dog lived for four hours, and then died. The above gentlemen have kindly allowed me to use their names as reference.

CHARLES UNDERWOOD.

January 1, 1851.

**SNAKE.**—On Friday last, a man in the employ of Mr. Gordon, of North West Bay, was lifting a log, when a Snake bit him in the fleshy part of his arm, and hung on it. The man shook it off, and wished his mate to cut a piece of flesh out of his arm; but he being afraid of injuring some of the blood vessels made but a small incision. He then made the best of his way to a medical gentleman, who was not at home,—and hearing that Mrs. Fisher had some of Underwood's Antidote, he went to her, and some of the medicine was applied. The man felt very sick for about two hours, but no other inconvenience resulted from the bite of the Snake. —H. T. Advertiser, Feb. 25, 1851.

**SNAKE ANTIDOTE.**—Another extraordinary cure has been effected by Underwood, upon the servant of Mr. Jessop, of Muddy Plains, who was severely bitten by a Snake a few days since. There can be scarcely a doubt of the efficacy of the Antidote, from the numerous respectable vouchers which he possesses. The public are, however, too prone to neglect these discoveries. We understand Underwood acquired his secret in the Brazil, and it is well-known how skillful the Indians of South America are in the cure of the bite of venomous reptiles. —Carmichael Chronicle, Nov. 29, 1851.

January, 1850.

I have to bring under the notice of the public that I, John Gorin, having been bit by a Snake on the leg, Underwood's Antidote being applied, life was spared.

Witness—T. CLARK,

Birnie's Bay.

J. C. WALL & Co., Printers, "Advertiser" office, Hobart Town.

## UNDERWOOD'S SNAKE ANTIDOTE.

To the Editors of the Advertiser.

DEAR SIR,—Between nine and ten o'clock on Sunday morning last, on the North West Bay Road, near Kingston, a little boy, aged about three years, walking barefooted, trod upon a large Black Snake, and was bitten on the sole of one foot. The child was immediately carried to the township, and taken into the cottage of Constable Riley, where Underwood's Antidote was freely applied to the wound, in presence of Mr. Chief District Constable Pittman, Mr. Keen of this place, myself and others. The child, until the application of the Antidote, appeared to be drowsy, and seemed to take no notice of the bite; but immediately on its application, he cried to have the staff taken away, and suffered such acute pain, that it was thought he was going into convulsions, and medical aid was sent for. The pain, however, gradually ceased in the course of the next few minutes, and after the lapse of about half-an-hour, he seemed quite recovered, and has continued so ever since.

The boy (who is an intelligent little fellow) in answer to an enquiry of mine, pointed out—while suffering from the pain of the Antidote—the place where he was bitten, and said "the Snake did it." The wound—which was a jagged one—also bore every appearance of being such as would be caused by the fangs of a large Snake.

I have every reason to believe that the application of the Antidote saved the child's life, and, I think, it only right that circumstances should be made known to the public, through the medium of the press, that the value of the preparation may be more appreciated by residents in the bush.

I would also further state, that some years ago, I applied the Antidote with success, to a finger of a female, which had been bitten by a Centipede. On that occasion, the finger had commenced swelling and became painful. The application of the Antidote caused a numbness, and after the lapse of an hour, nothing more was felt of the effects of the bite.

I am, dear Sir, yours faithfully,

E. INNES.

Police Office, Kingston, Nov. 7, 1853.

20th Jan., 1859.

To the Editors of the Hobart Town Advertiser.

MR. EDITOR,—Seeing an advertisement referring to Underwood's Antidote in your columns of this date, it has just jogged my memory,—and I think that it is rather neglecting on my part for not writing to you before about the matter,—if you could find space in your local to put it before the public.

At Stony Steps, about three weeks ago, one of my quartermen's wife was picking up some sticks and bark to light the fire with, by the side of a stump, when stooping, a Black Snake flew from the top of the stump, and bit her in the thick part of her arm. Fortunately I had bought a bottle of Underwood's Antidote from Mr. Millhouse some time previously, and left it in the hut in case of an accident. The men applied the Antidote, and the women was soon well a day or two after. My carter's dog was bitten in the foot, and it screamed fearfully, and I got two men to hold it until I ran to the hut for the Antidote; and in a very short time the dog was all right; and I should strongly recommend every person engaged in the bush to have some of this Antidote by them in case of an accident.

I am, Sir, yours, &c.  
JOHN GILLON,  
Stonemason, Macquarie-st.

hope that at last a veritable antidote for snake bite has been discovered.

## Underwood Passes into Folk-Lore.

It was reported in *The Argus* of November 6 and 12, 1861, that after careful investigation by the medical profession of Melbourne, it had been found that the antidotes of Underwood and Shires were inert. Also, in regard to a statement made by a witness, that he had once seen a snake bite an iguana, and that the latter animal partook at once of a particular herb which the observer imagined must be the antidote, it was stated that "it is now pretty generally believed that snake poison has no effect on a cold blooded animal".

Sixty years afterwards the same theme appears in *The Bulletin* (November 8, 1926), when "Dingo" wrote as follows:

Can anyone tell me what became of Underwood of snake bite fame, whose name was a household word along the N.S. Wales-Victorian border towards the end of last century? The bush folk there to a man believed in him, and claimed that he was the first white man to see the Goanna, after its battle with the tiger snake, nibble the mysterious life saving weed. He was also said to have been the hero of Banjo Paterson's poetical skit on the venerable tradition. In quite recent years what was claimed to be the last bottle of the cure was sold at Albury, N.S.W., in aid of charity and realised quite a decent sum.

A month later (December 6, 1926), in the same paper, "C.F." described Underwood's death as the result of having been bitten at Wattle Hill, Tasmania. He went on to say that some of the family had moved to Maoriland and still made the antidote; at least, long after his death, the family posted a bottle of it to his (C.F.'s) own father (who had known it, and had always kept some of the stuff in the house, never having had occasion to use it), asking that it should if possible be given a trial. Underwood used to say that the stuff from which the remedy was made was common, and people saw it every day. It has been hinted that bracken fern had something to do with it. The antidote was volatile and evaporated rapidly.

"G.I.H.", in the same issue, recalled how, when going to the Hutchins School at Hobart forty years previously, he had often passed a place in Argyle Street near the wharf, where Underwood lived. It was a queer house, some 20 feet below street level. Looking over the place one could see a small notice:

FIGURE III.—Instructions for the use of the antidote, and testimonials.

"Underwood's Antidote for Snake Bites." "G.I.H." mentioned this to his uncle, who said that the specific was made from dock weed. This weed is one of a dozen or more that have been mentioned as the vegetable which the goanna makes for after a snake bite.

It remained for "Banjo" Paterson to give lasting testimony to this widely held belief (Paterson, 1895). A year or two before his death, whilst on a visit to Hobart, he came to my home and looked over my books when we found that we had much to discuss with a common interest in Australian literature. At that time I did not know that his famous work, "The Man from Snowy River", included a ballad "Johnson's Antidote", which must have spread the goanna legend far and wide. Here is a portion of one of its verses:

Johnson was a free selector, and his brains went rather queer,

For the constant sight of serpents, filled him with a deadly fear;

So he, tramped his free selection, morning, afternoon and night,

Seeking for some great specific that would cure the serpent's bite . . .

He sought out King Billy, of the Mokka, who on being questioned, spoke of the goanna's curing himself by eating "little pfella tree":

That's the cure, said William Johnson, point me out this plant sublime,

But King Billy, feeling lazy, said he'd go another time.

Thus it came to pass that Johnson, having got the tale by rote,

Followed every stray goanna, seeking for the antidote. The remainder of the poem and its dénouement will be easily found by those interested. It is sure that Paterson's early up-country life and long association with the outback had made him familiar with the claims of such "cures".

#### Conclusion.

Much of this long narrative was written at Oyster Cove, on D'Entrecasteaux Channel, where my family has had a second home for almost a century. Here most of our school holidays were passed, and we had been well instructed as to what to do in the event of snake bite. Eventually when my sister was struck by a black snake, local treatment and a ligature were effective. On another occasion a young girl from further up the gully was brought down for help. At that time I was a fifth year medical student; similar measures ensured her recovery, and gave me a small amount of transient fame. In both instances the bites were on a finger.

On a hot summer morning last February my brother-in-law came in to say that he had just killed a large tiger snake in front of the house. That afternoon an elderly friend, who lives two miles away, came in, and during afternoon tea, it occurred to me to ask her whether she had ever known of Underwood's remedy being used locally. Incidentally, a family of that name and reputed to be related to Underwood still lives some five miles from our house, back in the hills. She replied that she had not, but that she had heard of Underwood's actually witnessing an encounter between a snake and a goanna; the latter, having been struck, moved into the bush and was seen to eat leaves from a low bush. She added that it was from this bush that the remedy was prepared. So, it seems, the traditional belief still remains. In southern Tasmania. All that has been written above goes to show that in spite of adverse reports from the scientific experts of their day, Underwood's antidote had a wide circulation in Tasmania and to a lesser extent Victoria and New South Wales. In our district, one of small fruit pickers, the fear of the effects of snake bite is as real today as it was in the period under consideration. So it may be readily understood what a real sense of help and security such antidotes afforded in the period under consideration, to those whose lives were passed in remote areas of the island, far from immediate medical assistance.

#### Acknowledgements.

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#### CHANGING OF DISEASE PATTERNS: DISAPPEARANCE OF DISEASES: NEW DISEASES.

By CHARLES ENGEL,  
Sydney.

THE more time advances, the more I am puzzled and perplexed by the enigma of chlorosis. Once a well-known, common disease, it has become progressively less frequent since about the turn of the present century; more precisely according to Campbell (1923), since 1903. Now it is rarely seen in any part of the world—some authors even state that it is never seen.

Chlorosis was a special hypochromic anemia, almost invariably affecting young girls in adolescence, between the ages of fourteen and twenty years, very rarely earlier and seldom later, with a distinctly greenish tinge; hence its name (*χλωρός*, green). Its popular designation was "the green sickness". It was first classically described by Johannes Lange in 1554 as "morbus virgineus" (Major, 1939), but it was certainly well known much earlier. It was so conspicuous and so interestingly characteristic that "it was portrayed on canvas by many famous artists of the seventeenth century" (Fowler, 1949). It has been regarded as a well-defined clinical entity. It often attacked several members of the same family. Its cause and pathogenesis were never elucidated. Poor and improper food, overworking, confinement to badly lighted rooms, lack of exercise, want of fresh air, and wearing of tight corsets were incriminated, but no convincing proof has been presented for any of the supposed causes. It certainly occurred mostly in girls who lived under unhygienic conditions. It was extremely common in domestic servants and factory workers, but it was not rare in girls of the wealthiest families. It must have been an iron-deficiency disease, as it responded almost without exception promptly to iron medication. However, recurrences of chlorotic attacks were not rare even comparatively late in life.

That it must once have been very common is well demonstrated by the fact that eminent and critical experts, authors of monographs dealing with the disease, could report hundreds of cases. Bramwell reviewed 314 cases in 1899, and von Noorden 217 cases in 1905. Sir Robert Young wrote as follows in 1950:

Chlorosis was a very common condition in my early years of practice. At out-patients on a Saturday afternoon I often used to see up to four or five new cases and, of course, corresponding numbers of old ones.

Let us quote some data about the frequency of occurrence of chlorosis from hospital statistics. In the out-patient department of the St. George Hospital in Hamburg there were 201 cases of chlorosis among 12,000 admissions in 1901; in 1923 there were only three cases among 20,000 admissions (von Hoesslin, 1926). In the out-patient



department of the Massachusetts General Hospital in the period from 1903 to 1926 there were 115 cases of chlorosis among the first 50,000 admissions; among the eleventh 50,000 patients only one single case was encountered (McCrae, 1927). However, I hasten to add here that, according to the opinion of some authors, chlorosis was formerly diagnosed too often, and so probably was not rarely misdiagnosed. Many cases might have been due to latent tuberculosis of the lungs and latently bleeding gastric ulcers, X rays being at that time not generally available for the normal routine examination of patients.

We find already in the book on hematology by Gulland and Goodall (1914) that "There seems to be little doubt that chlorosis is becoming a less frequent condition than formerly". Since that time similar statements are to be found in hematological treatises and in text-books of diagnostics and of internal medicine in languages of nearly all civilized countries. "The disease seems to be dying out" (McCrae, 1926). "Chlorosis, or the green sickness, used to be one of the commonest of diseases; now it has become remarkably rare, and is one of the best examples of a disappearing disease" (Boyd, 1950). "Chlorosis of which a lucid description has been given by Hoffmann over two centuries ago and which was to judge by the literature a common disease early in the century was gradually to die out as the years advanced" (Cohen, 1950). "Since the end of the nineteenth century its incidence rapidly decreased until at the present time it is extremely rare in its classical form" (Fowler, 1949). "It is now of little more than historical interest" (Dible, 1950). "In the form defined above the condition has almost ceased to exist" (Whitby and Britton, 1953). In the two-volume "Medicine" of Garland and Phillips (1953) it is mentioned in the introductory chapter that chlorosis has disappeared; in the chapter on diseases of the blood we do not find a single word about it. In some recent similar books chlorosis is not even mentioned.

But this apparently general consensus has not been allowed to go unchallenged. There are opponents too: "The disappearance of chlorosis has perhaps been exaggerated" (Wintrobe, 1951). "Actually chlorosis is not a rare disease" (Castle, 1950).

I definitely do not agree that the disease may still exist, but under another name. The idiopathic hypochromic (achlorhydric) anemia of today occurs at a much more advanced age—in women aged between thirty and fifty years—is of longer standing, and is, in striking contrast to chlorosis, notoriously much more resistant to treatment than chlorosis was. I would not make this apparently superfluous remark had this disease not been termed by some authors "chronic chlorosis". I feel sure this term is an absolute misnomer and would be better abandoned. It is not fitting at all. These patients never show the characteristic greenish tinge of real chlorosis. Moreover, in chlorosis fairly normal gastric secretion has been found and very often even hyperchlorhydria. Misleading also is the term "tropical chlorosis" or "Egyptian chlorosis" (anemia of young girls in the tropics suffering from hookworm disease), and I think it should not be applied either.

I myself find it very remarkable that, whatever may have been the cause, chlorosis has not reappeared in the stormy days of the present century, during and after the first and especially the second World Wars in countries where people were near starvation and living under the worst possible conditions of hygiene. Nor could any manifestation of chlorosis be detected in the dreadfully malnourished girls in the miserable forced labour camps of the second World War, where the iron intake must have been practically nil.

To sum up: chlorosis has become progressively more rare since about the beginning of the present century until it has virtually disappeared. Its cause and pathogenesis were unknown, and in the same way we can find no explanation for the disappearance of the disease. Chlorosis was never clearly understood. It was a mystery, and in its disappearance it has become an even greater mystery.

I have to confess that I feel somewhat ashamed and humiliated that we cannot cope with such a seemingly simple problem. We have every reason to be highly elated at and very proud of the stupendous progress medicine has made in every field over the last fifty years. "Medicine of today is entitled to be loud with jubilant terms of victory." However, the necessity of admitting such a failure is a strong warning that, in spite of everything, we have to remain modest and humble and free of pride.

Does it happen often that a disease disappears all over the world? On the contrary, it happens rarely. There were mass psychic epidemics in many countries of Europe in the Middle Ages (dancing mania—Saint Vitus's or Saint John's dance; flagellantism—contagion of penitential self-scourging with leather thongs) which disappeared completely; but, of course, these were no real disease entities. There was ergotism—chronic ergot poisoning. There were two forms of it: the gangrenous form, with progressive moist or dry gangrene of the fingers, or more often of the toes, and the spasmodic form with nervous symptoms—itching, tingling, loss of cutaneous sensitivity, amblyopia *et cetera*. It was once common. There were quite severe epidemics of it in parts of Europe. It disappeared almost completely with better methods of grain handling (Koppányi, 1936). Often an infectious disease or a nutritional deficiency disease disappears in a country, in a block of countries, on a continent (for example, typhoid fever in Australia) or even all over the world, as we shall see later on; but there is no parallel in human pathology for the disappearance of such a "constitutional" disease as chlorosis.

I feel sure that it is worth while to go into the details of the subject of "changing of disease patterns, disappearance of diseases, new diseases". In my opinion, the subject is of great importance and deserves much thought. It is likely that any one of us may be confronted at any moment with embarrassing problems in this field. But more: there is to me little room for doubt that many members of the younger generation of the medical profession will experience many surprising and, even at the present time, unimaginable changes in this field. We have to look into the past and study the present so that we shall always be on guard and quick to perceive the change in the future.

The subject is a vast one. I have to restrict myself to the most important items. The reader will find an unusually large number of quotations. I think them advisable, to show clearly that the given statements and data are collected from the experience of a great many first-class experts and not only from my own experience.

Can the statement of Charcot—surely the most eminent and famous French clinician and medical scientist of the end of the last century—be upheld? It was as follows: "Disease is from old and nothing about it has changed. It is we who change as we learn to recognize what was formerly imperceptible." I found these words in the introduction to Garrison's "History of Medicine" (1929), in which a long series of the most noteworthy medical sayings are collected. There is surely an element of truth in the saying.

Mumps and tetanus appear today in the same form as that described in such a masterly way by Hippocrates. Smallpox in its classical and repulsive form is occasionally still to be seen in the east, and there are years in which thousands die from it (epidemics among pilgrims in Arabia). Indeed, nothing has changed about these diseases. Coronary infarction has surely occurred constantly over the last centuries; but as a well-defined clinical entity it was recognized and accepted only about three decades ago. In this case, indeed, we have changed and not the disease. The same holds for severe sciatica. Over many centuries extremely painful sciatica (described as Cotugno's disease in 1765—Major, 1939) was known and had been treated everywhere. But only in recent times, about twenty years ago, was it recognized that in a large proportion of these cases the condition is due to a pro-

lapsed disk and can be cured by surgical intervention. Again, we have changed and not the disease. "Glenard's disease (visceroptosis) has not disappeared: it is merely not diagnosed any more" (Garland and Phillips, 1953). The same holds true for splenic, secondary anaemia and *icterus catarrhalis*. Once more we have changed and not the diseases. A large number of similar examples could be collected.

But, in spite of all that, I would not hesitate to assert that, to me, just the reverse of Charcot's saying seems much more plausible at the present time. I would even go so far as to deny it completely. Diseases and the patterns of diseases have changed in the past, are changing very much at the present time, and will change in the future to an even greater extent. Medicine is in motion, and the changing of diseases is in steady motion. The two and a half thousand years old basic dictum of the Greek philosophy *panta rhei* (Heraclitus), "everything is moving", well applies to the changing pattern of diseases. Or as Heckel, the famous German biologist, said about a hundred years ago: "*Nichts ist beständig, bloss der Wechsel*" ("Nothing is constant, but change"). "And never before has the constantly changing character of diseases become so apparent as today" (Boyd, 1953). I hasten to add that the tempo of change is also incomparably faster than it has ever been before. Progress achieved in medicine has brought about the change. It is the consequence of modern civilization with its prophylactic sanitary measures and efficient active therapy with newly invented drugs. Let me repeat: diseases and the patterns of diseases change and must change constantly. That this is so can easily be deduced and proved by analysis of the meaning of "disease".

"Diseases do not exist as natural phenomena like plants and animals to be discovered. Indeed they are never discovered. They are created by man as conceptual symbols and he may later discard them. Disease is not an entity any more than are the minutes and hours of the astronomer or the meridian lines of the navigator. Disease is not a state, it is rather a process ever changing in its manifestations" (Garland and Phillips, 1953).

Disease is the reaction of (i) an individual (ii) to a given cause (iii) under special circumstances. The sum of the reaction is defined as disease for the purpose of describing it, so that it can be dealt with in words and letters, that it can be taught and learned. (i) Individuals are different in age and race, in habits, in nutrition, in resistance (disposition and immunity), in psychosomatic conditions. (ii) The cause of the disease is different in any case, in quality, in quantity and in intensity. (iii) The circumstances—living conditions, surroundings, climate, food—always differ to a very large extent. It is therefore only natural that the reaction is always a different one, and that there cannot and does not exist a rigid pattern of any disease. No two individuals react to the causes of any disease in exactly the same way. No two cases are ever quite the same. Every case of a special disease gives a new pattern. The text-books outline the approximate average of a disease, delineating the cardinal features of the normal picture and the normal course. But they describe every deviation from these in detail—the abortive and light cases, the severe cases, the complications which produce an abnormal course, the fatal cases. All these varieties of clinical picture may occur within an epidemic even in the diseases of one and the same family. It may happen, and has happened quite often, that four siblings acquire poliomyelitis in the same house from the same source. Every one of the patients may show a different picture, from the mildest to the most severe. Identical twins living under the same conditions in the same flat may present a different pattern of disease originating from the same source. The pattern of one and the same disease varies even with the same person at different times. Almost every attack of rheumatic fever shows a different pattern. It is well known that there were, and still are, people who have a special susceptibility to erysipelas or pneumococcal pneumonia. Both diseases may have occurred in the same individual up to ten times or even more often.

Well nigh every attack differs in its clinical picture even when the erysipelas starts from the same area of the skin. The extent and severity of the pneumonia vary a good deal even when it occurs within some years.

"Infectious disease is constantly changing its pattern" (Burnet, 1953). Every big wave of an epidemic had in the past and has at the present time its own "*genius epidemicus*". Every physician of the older generation has vividly retained the memory of the terrible influenza epidemic of the years 1918 to 1920, with the rapidly progressive confluent pneumonia of both lungs, and the picture of terrific sudden vasomotor collapse, and the horrifying mortality. The pattern of the disease was quite different from that of any epidemic observed until that time or since that time.

#### Changing of Disease Patterns.

The changing of disease patterns is most spectacular in the realm of the infectious diseases and of the nutritional deficiency diseases. I would even state that the change in these fields is sometimes revolutionary.

The improvement in social and economic conditions (less slave working, less poverty, better agriculture, and hence more and better food), in environmental conditions and in effective sanitation (better housing, better sewerage, supply of safe, good water), in preventive medicine (vaccination), and in education has brought about a definite decrease of most of the infectious diseases, and with it a change in their pattern.

By the foregoing measures the fulminating and catastrophic epidemics of the past have been almost completely eliminated. However, small or large waves of epidemics are still seen even today in one or other part of the globe (see World Health Organization Reports, 1954). Some of them are levying a toll of hundreds or thousands, even of hundreds of thousands, somewhere on the earth (the victims of malaria still amount to about 3,000,000 a year), but the incidence and number of deaths from all of them are almost without exception smaller than they were some decades ago, except perhaps for poliomyelitis and hepatitis epidemica.

The difference in the present pattern from that of the past is in some infectious diseases enormous, in others less conspicuous.

The most spectacular and surprising change in the character of an infectious disease is that of scarlet fever since the beginning of the century, and to a somewhat less extent that of diphtheria. "Twenty years ago diphtheria and scarlet fever patients would occupy four-fifths of the beds in any fever hospital. These cases have now shrunk to a mere fraction of their former number. A fewer number of beds are required in fever hospitals mainly because of two factors: (a) the virtual disappearance of diphtheria and (b) the lessened need for admission to hospital of scarlet fever patients" (McLorinan, 1954). This is so obviously because the severity of scarlet fever has lessened very much and it is therefore no longer alarming to the parents. The toxic or malignant type of scarlet fever is exceptional today. Cases of post-scarlatinic glomerulonephritis are much rarer nowadays and much milder than they were previously. In the last fifty years the mortality rate from scarlet fever has dropped from 13.5% to less than 1% (Sheldon, 1951); according to some authors, in some countries it has dropped nearly to zero. In the United States of America many State and local health departments have abandoned quarantine of scarlet fever patients (Ceeli and Loeb, 1951). According to the World Health Organization Report (1954) there were 4977 deaths from scarlet fever in the United Kingdom in 1901. In 1952 there were 25 (90 altogether with the deaths from streptococcal angina). Three deaths notified from scarlet fever were notified in Australia for the year 1952, and 33 from diphtheria. Intubation and tracheotomy, often required in the treatment of laryngeal diphtheria some decades ago, are now rarities. At the Prince Henry Hospital, Sydney, intubation has not been carried out in any case of laryngeal diphtheria for several years, and only



three tracheotomies were performed from the end of the year 1946 until the beginning of the year 1953.<sup>1</sup>

I will discuss now in due order three important diseases whose pattern has altered a great deal in the present century. These are typhoid fever, pneumococcal pneumonia and erysipelas.

"Typhoid fever is no longer the scourge of yesterday." Boyd (1950) writes as follows: "The clinical picture seen in Winnipeg and in the Province of Manitoba has altered considerably." The same holds for nearly all civilized countries; the disease has become much rarer and milder. In some less civilized countries the morbidity and mortality from it are still high (World Health Organization Report).

In countries where the malady has become rarer and milder, the typhoid stage from which the disease's name is derived (*typhos*, cloud) with its mental torpor and dulness and continuous high delirium going on for weeks, is much less frequently seen at the present time. Even rarer are perforation of a typhoid ulcer and life-endangering severe intestinal hemorrhages. "With continuation in improving sanitation as to food, water and sewerage and in isolation techniques in caring for typhoid patients eventually typhus will become a disease that students of medicine and physicians will know only from reading" (Christian, 1947).

The second disease which has lost its frequency and severity to a very large extent is pneumococcal pneumonia. At the beginning of the century pneumococcal pneumonia was one of the most widespread and acute infectious diseases. "At the present time pneumococcal pneumonia is not a frequently observed disease" (Christian, 1947). The text-book pattern—initial chill, continuous high fever for about a week, and then the classical crisis—is not so regularly seen as before. The complications of pneumococcal pneumonia, purulent pleuritis, pericarditis, meningitis, arthritis—are definitely less frequent than they were at the beginning of the century. Pneumococcal pneumonia was for centuries one of the most dreaded diseases—the "captain of the men of death". The mortality from it has dropped everywhere to a large extent; some authors estimate the drop at 50%, others believe that the fall in mortality is even greater. Six hundred and seventy deaths from lobar pneumonia were notified in the year 1952 in Australia; I think that probably only a small fraction of these were due to pneumococcal infection.

Erysipelas appears in a greatly mitigated form at the present time in comparison with its previous severity. "On the whole erysipelas has been decreasing in recent years" (Christian, 1947). The severe forms of this disease (very high fever, extensive inflammation and signs of toxæmia, involvement of the deeper tissues) are much less frequent nowadays than they were. Only exceptionally can be seen the "migratory form", in which the inflammation slowly creeps over the body for weeks and weeks.

These changes are mostly spontaneous and their origin is not fully understood. They are first of all due to increased efficiency of hygienic measures. Very likely, however, they have been produced by the attenuation of the virulence and invasive power of the provocative agents. However, the most striking change in the pattern of these three diseases has occurred in the last ten to fifteen years, as a result of the therapeutic revolution accomplished by the use of chemotherapy (sulphonamide drugs) and of the antibiotics. Many patients with these once ominous diseases respond so promptly to modern treatment that they have become comparatively trivial.

For a large number of these diseases I would agree with the suggestion that I have read somewhere, that the old Latin saying "*medicus curat, natura sanat*" should be changed into "*medicus sanat*".

In pneumococcal pneumonia the fever abates mostly within twenty-four to forty-eight hours, with a dramatic change for the better. Most of the patients can be regarded

as cured within a couple of days. Very interesting are the statistics of Cannon (1955):

From 1927 until 1940 in a series of 2000 consecutive necropsies lobar pneumonia was considered to be the principal cause of death in fifty instances. Since 1940 in a succeeding series of 2000 consecutive necropsies the total number of deaths attributed to lobar pneumonia was only seven and for the past few years we have had no cases at all. Our residents and medical students now learn about the pathological aspect of pneumonia only from books, from the reminiscence of older clinicians and pathologists and from museum specimens.

With "Chloromycetin" the continuous high temperature of typhoid fever steadily subsides to normal within a period of days in a very large proportion of the cases, though relapses are not rare. In erysipelas the whole disease is mostly completely controlled within forty-eight to seventy-two hours, with progressive recovery.

The beneficial influence of modern therapy is very striking in cerebro-spinal fever too. In earlier times its mortality rate in very severe epidemics was up to 80%. Today relatively few patients die from it, and hardly any nervous sequelæ of the disease are encountered if active therapy is initiated at the earliest possible stage.

The picture of gonorrhœa has altered also to a very large extent. Active therapy is so efficient that it can be cured completely within some days, almost without exception. Complications from it can probably be prevented if treatment starts—I use the same words again—at the earliest possible stage. The crippling lifelong diseases of the female genitalia as well as gonorrhœic arthritis have become extremely rare.

The pattern of acute rheumatic fever has changed very much since about the end of the last century. On the one hand, the incidence has lessened to a very large extent, and on the other hand the disease has become much milder. Copeman (1946) puts it as follows:

In olden times the most striking features of the disease were: high pyrexia, excessive sweating and peracute, very painful inflammation of the joints. The inflammation jumped over from a couple of joints to others, mostly in pairs, and this went on and on over many weeks. At the present time it is rare for the temperature to exceed 102-103, whilst the arthritis is in most cases of short duration and of very moderate severity.

It is interesting to note, however, that in spite of all that, the endocardium does not appear to be less frequently involved. The old French saying was as follows: "Rheumatic fever licks the joints, but bites the heart." And today? Rheumatic fever still bites the heart, but licks the joints less. Nevertheless the incidence of rheumatic heart disease has dropped very much in the last thirty years. Stroud (1954) writes as follows:

Certainly the incidence of rheumatic fever and rheumatic heart disease is much less. Personally, my experience at the Children's Heart Hospital in Philadelphia which was started some twenty-eight years ago proves this fact, since from its inception with the hospitals of Philadelphia referring children with active rheumatic heart disease for convalescent cure to the institution we had a constant waiting list of over 250, whereas in the past few years we had no waiting list at all.

The mortality from rheumatic heart disease has fallen dramatically since the beginning of the century (Arnott, 1954), undoubtedly owing to the much lessened incidence of rheumatic fever, and to the fact that recurrence of attacks of rheumatic fever can be successfully prevented, or at least considerably lessened, by continued chemoprophylaxis.

Rantz's (1954) opinion is that rheumatic valvular heart disease will be uncommon in children who grow up under optimal circumstances during the next twenty years. I definitely agree with the statement. Lessening of poverty decreased in the past and will decrease the incidence of rheumatic fever even more in the future. "The disease is supposed to be 20 or 30 times more frequent among the poor than among the well-to-do" (Lichtwitz, 1944).

<sup>1</sup> I have to thank Dr. C. I. W. Walters, General Medical Superintendent of the Prince Henry Hospital, for this information.

The involvement of the brain from rheumatic fever (rheumatic meningocephalitis) occurs almost certainly less frequently than before. Severe cases of chorea as sequelae of rheumatic fever are now relatively rare.

Much changed also is the clinical picture of syphilis over the last century. The once violent epidemics of the disease have ceased completely. Alteration of the common pattern of the disease has been observed and described already in the first decades of the present century. The formerly hideous and destructively ulcerated gummata of the skin and of the mucous membranes (nose, throat) and the gummatous ulcerations of the bones have disappeared almost completely. Children or adults with saddle nose (gummatous ulceration of its bones) are rarely seen today, nor are large perforations in the palate. The recent treatment with penicillin produces such a rapid, beneficial effect when initiated in the early stage that even the secondary skin and mucous membrane manifestations appear today in a much milder form, if they appear at all. Much rarer at the present time are the severe forms of aortic aneurysms (bulging out on the surface of the chest, compression of the trachea and of the bronchi, external rupture of these aneurysms *et cetera*). But simple aneurysms are less frequent too. "Between the years 1912-1916 I saw a great number of ward beds in the hospitals filled with patients with syphilitic aortic aneurysms" (Stroud, 1954). Today there are months when no patient with syphilitic aortic aneurysm can be seen in the wards of any big hospital.

Almost certainly the malignant, rapidly progressing forms of cardio-vascular syphilis are less common too. Syphilis of the liver is rarer. "Syphilis of the liver has sunk now to the obscurity of small type" (Boyd, 1953). All kinds of cerebral and cerebro-spinal syphilis are much less common than they were before; it appears that this type of syphilis has declined even more than syphilitic aortitis. Fewer and rather milder cases of tabes and of general paralysis are seen nowadays than in former decades, and the mortality from them is progressively decreasing. In Australia, general paralysis of the insane was notified as the cause of 138 deaths in 1915, but of only 20 in 1952. Charcot's disease of the joints is less common. The number of cases of severe congenital syphilis is very small today. "No instance of congenital syphilis was recorded in 2743 necropsies in children less than ten years of age during the years from 1949 to 1953" (Cannon, 1955). Some authors believe that syphilis is dying out and will completely disappear within a couple of decades. I agree with them. It does not seem to me a vain Utopian belief, in spite of the fact that I have gained the impression in the last two years that the control of the disease will prove to be a much harder task than had been supposed. It is very important from this point of view that the *Spirocheta pallida* has so far not developed any immunity against penicillin. I believe that from the above-mentioned viewpoint it is a good omen that "Venereal diseases have now become so much less troublesome as causes of morbidity and mortality than they formerly were that too few papers are being written to sustain the *American Journal of Syphilis, Gonorrhea and Venereal Diseases*, so that with its thirty-eighth volume the journal is brought to a close" (annotation, *Brit. M. J.*, December 4, 1954).

In the field of tuberculosis the change is much less spectacular, but there is surely a distinct change in its pattern too. "As a result of the campaign against tuberculosis a generation of children and young adults have grown up which have escaped primary infection and are negative to tuberculosis. This has produced a great change in the disease pattern" (Boyd, 1950). As a cause of death tuberculosis has dropped considerably in many countries (Scandinavia, United States of America, United Kingdom); in other countries the decrease is less extensive. The once severe cases of tuberculosis of the bones (spine) and joints, and of abdominal tuberculosis are almost certainly less common and of a milder type today. Once, about 50% of young diabetics died from tuberculosis; at the present time this combination is an exceptional rarity. Fewer cases of Addison's disease are due to tuberculosis than

before. A stupendous change has been achieved by modern treatment in the formerly depressing clinical picture of military tuberculosis and especially in meningeal tuberculosis. A large number of children afflicted by these diseases can be completely cured nowadays by combined active and prolonged therapy. "It is an encouraging thought, that given the necessary money and enthusiasm modern methods could eliminate tuberculosis from any community within a generation" (Burnet, 1953).

Diseases which have also become much milder, owing both to better hygiene and to therapy, are puerperal fever, acute osteomyelitis, *mastoiditis purulenta*, inflammation of the nasal sinuses, *meningitis purulenta*, hemolytic streptococcal angina and the different kinds of septicopyemias. There is also a host of other diseases, once widespread and ominous, which have lost much of their former frequency and often catastrophic severity (typhus, cholera, yellow fever *et cetera*).

Thanks to efficient sanitation measures, hookworm disease, once one of the most prevalent diseases in the world, has become less frequent and less severe in civilized countries.

Very interesting is the changing of the pattern of sub-acute bacterial endocarditis. Formerly it was regarded as a uniformly malignant, practically hopeless disease. Patients were bedridden and suffering for many months, even for one or two years. Death was inevitable. At the present time the outlook is much more favourable, as it is well known that a large number of patients (up to 80% and even more) can be cured with very large doses of penicillin and other antibiotics. In many cases, of course, heart failure remains.

To sum up: The pattern of infectious diseases has changed considerably in the last half-century. We have seen that many of them became much milder than they were before. "Unless something unexpected happens there will be soon little need for large permanent hospitals for infectious diseases" (Southwood, 1955). It is certainly no idle optimism to expect that the change will go on. It is certain that every effort will be made to ensure that the conquest of infectious diseases is virtually complete. If so, it can be hoped with justifiable optimism that some of the diseases caused by bacteria and spirochaetae may be eradicated in the not too distant future, so that they will disappear completely and will be only of historic interest.

Very much changed is the picture of nutritional deficiency diseases. In the far east there are still countries (Japan, China, the Philippines) where, for example, beriberi takes a big toll of victims. In the more civilized countries of the west, frank and severe cases of scurvy and of beriberi—perhaps not so much of pellagra—are much less frequent than they were, though heart disease caused by thiamine deficiency occurs occasionally in the western hemisphere and in Australia too. Scurvy has disappeared from the naval services. It is important that in recent years pellagra has become much milder in the United States of America, where the disease claimed many thousands of victims even some years ago. Severe and crippling cases of rickets are very rare today in civilized countries all over the world. There is growing evidence that, with the lessening of ignorance and of poverty, with the increase of knowledge and with the supply of more and proper food, the frequency of severe cases of nutritional deficiency diseases is slowly lessening in semi-civilized tropical countries too.

As a consequence of the lessened incidence of protracted severe purulent conditions of bones (chronic osteomyelitis, syphilitic and tuberculous breaking-down of the bones and of the joints), chronic empyema, chronic lung abscess *et cetera*, amyloidosis has become a rather rare disease. "Fortunately the once common and usually irremediable problem of amyloidosis is a vanishing one" (Fishberg, 1954).

Some authors believe that gout is on the decline; others deny this. However, it seems very probable that fewer advanced cases are encountered today than for some decades.



Very much changed is the picture of patients with congestive heart failure. Members of the older generation retain vividly the memory of patients with intractable oedema of the legs, the buttocks, the lower part of the back and the abdomen, which transformed the unfortunate patient into a distorted, helpless mass. Bilateral hydrothorax and hydroperticardium and ascites produced a persistent, distressing dyspnoea. The advent of the mercurial diuretics completely transformed the picture. At the present time, widespread oedema from congestive heart failure is hardly ever seen. There are young doctors who have never seen such patients, and even the pathologist only exceptionally sees on the post-mortem table a subject with widespread oedema. Nowadays patients with heart failure never succumb to generalized oedemata.

There are many other diseases which have changed considerably or are progressively changing at the present time. Pernicious anaemia is not pernicious any more; young diabetics are not so thin and emaciated as they once were; hypothyroid patients have no longer broad and coarse features. "The incidence of any kind of glomerulonephritis has decreased all over the world" (Fishberg, 1954). Not only does lack of space preclude a discussion of all these diseases here, but it would not even be timely and interesting any more, as they are so well known.

That neuroses can change their pattern very much is clearly demonstrated by experience in the two World Wars. In the first war the pattern of war neurosis was trembling and shaking of the whole body with pseudoparalysis; in the second World War heart and stomach troubles were the main complaints.

It is very interesting that the pattern of hereditary diseases has not changed over the last half-century. It is rather a surprise to me. Mutation of genes responsible for hereditary diseases occurs without doubt very frequently. "The general rule for organisms is that a change in character through mutation is much more likely to be injurious than beneficial" (Colin, 1946). One would therefore expect a long list of variations and new combinations of hereditary diseases. However, I have not been able to find any mention of a changed pattern of such diseases in the recent literature. Experts are definitely of the opinion that the pattern has not changed and that the hereditary diseases have not become more frequent. It seems that mutation of genes is so far on the whole mostly inefficient in mankind and does not cause any conspicuous effect. It seems, furthermore, that the inheritance of abnormal genes as the cause of hereditary diseases is well counterbalanced by the fact that a certain number of affected persons die early and the fertility of others is greatly reduced. It is even the opinion of Lenz (1931) that the incidence of hereditary diseases will rather lessen in the future as a consequence of lessened inbreeding caused by the modern facilities of international transport.

#### Disappearance of Diseases.

The changing of a disease pattern reaches its maximum when the disease disappears.

Burnet (1953) describes the spontaneous disappearance of three virus diseases as follows. The first was the "sweating disease" of the Tudor period, the "English sweat" of continental authors. It seemed to come to England in 1485, became an acute fatal epidemic and vanished completely after 1526. The second is *encephalitis lethargica*, which came from Roumania in 1916 and spread in the next seven years all over the world in pandemic form. In 1923, cases began to occur less frequently, and no certain case has been reported since 1930. The third example described by Burnet as a mere local and less important disease is the Australian X-disease, which appeared in 1917-1918 as acute encephalitis and disappeared in 1925. Burnet believes that "similar episodes will doubtless be as characteristic of the future as of the past".

It is the opinion of some authors that *prurigo ferox* Hebra is disappearing. In a discussion on the subject Rothman (1954) makes the statements that he has not seen it since the middle of the twenties and that he did

not see a single case of it in the last fifteen years in the United States of America. F. Goldschlag informs me that it was in 1918 that he saw his last patient with *prurigo ferox* Hebra in Vienna; he has seen none since that time. The disease was first described in Vienna and was formerly seen chiefly in Austria. I therefore made an inquiry about the disease at the Dermatological Clinic of Vienna. Professor I. Tappeiner was kind enough to report to me that not a single case of the disease has been seen in the clinic for more than two decades. He believes that the disease has virtually disappeared.

The once rightly dreaded *gangrena nosocomiale* (hospital gangrene), a contagious gangrene arising in crowded conditions where there was an absence of antiseptics, has almost completely disappeared. Fortunately the similarly dreaded hospital infection of wounds of the pre-Listerian era is also disappearing. Much rarer nowadays are the once fairly common deeply penetrating and sacrum-involving pressure bedsores (*decubitus*). Similarly, a very rare disease today in civilized countries is noma (a progressive gangrene of the buccal mucosa resulting in a perforating ulcer of the cheek).

#### New Diseases.

Entering now the field of new diseases, I have first to clarify the meaning of a "new" disease. According to the definition previously given, disease is the reaction of an individual to a given cause under special circumstances. From our present point of view the emphasis has to be put on "the cause of the disease". If a new agent so far unknown as the cause of a sickness produces a malady, it is a "new disease". If a hitherto unknown chemical substance or virus caused pneumonia, it would be a "new disease". If a substance so far unknown as a specific provocative agent produced allergic reactions, asthma, agranulocytosis, aplastic anaemia or arteritis, it would be a "new disease", even if in the text-books it was dealt with in the respective chapters.

In this sense I have to deal here in the first place with the wholesale change of the normal flora in the bowels and with the upsetting of the ecological balance in them called forth by antibiotics ("Aureomycin", "Terramycin", tetracycline, and less by penicillin and streptomycin).

The *Bacterium coli* especially disappears very quickly, being replaced in the first place by *Proteus vulgaris*, but occasionally by streptococci and staphylococci too. The bacteriological change in the bowels may be the cause of vitamin deficiencies (sore mouth and throat, rhagades in the corner of the mouth, cheilosis, glossitis with black hairy tongue) and "bizarre, new infections" (Beckman, 1954). In most cases the symptoms are simply slight or moderately severe diarrhoea followed by perianal pruritus, pain and bleeding on defaecation. In other cases very severe enterocolitis develops, of a pseudomembranous character (Reiner *et alii*, 1952), which proves very obstinate and resistant to treatment and has a critical tendency to relapse. Unfortunately many cases have been described of cholera-like staphylococcal enteritis which proved intractable and became fatal. Such enteritis is particularly serious as a post-operative complication when antibiotics have been used. Speare (1954) reviewed 23 such cases, 19 of which were fatal. Boyd's (1953) term for this disease is "antibiotic enteritis". The use of antibiotics may similarly become the cause of systemic monilial infections. "There can be no reasonable doubt that systemic monilial infections are much more common than previously and that they are induced in some way by antibiotic therapy" (Beeson, 1954). There have even been described cases of fatal generalized moniliasis and aspergillosis (Kligman, 1952). Much has been written about the allergy-producing effect of antibiotics, especially of penicillin, less so of streptomycin, "Chloromycetin" and the other antibiotics. They cause skin diseases (urticaria, dermatitis) and systemic patterns. They may produce very serious symptoms, anaphylactic shock and unfortunately fatalities too, especially after treatment with penicillin. "Fifteen deaths were reported in an 18-month period during 1952-1953" (Kagan and Fuller, 1955).

Earnest warnings have been given by experts of high standing against the indiscriminate use of antibiotics.

A new disease is vertigo and deafness caused by the neurotoxic effect of streptomycin and dihydrostreptomycin on the acoustic nerve. It is interesting furthermore that drug encephalitis, formerly known only after the use of arsphenamine and sulphonamide drugs, is now apparently produced by long-continued treatment with streptomycin (Hannicut *et alii*, 1948) and with para-aminosalicylic acid (Boyd, 1953). It is almost generally agreed that the higher incidence of arteritis and *polyarteritis nodosa* is due to the allergic effect of sulphonamide drugs and of antibiotics.

It seems that epidemic hemorrhagic fever of Korea is a new disease. It broke out in the autumn of 1951 and continued throughout the winter of 1951-1952. The aetiology and epidemiology remain a mystery.

Much more frequent than they were before are the severe alterations in the bone marrow producing agranulocytosis and aplastic anemia. These conditions have come much to the fore in recent years. They are caused by the greatly increased consumption of drugs with partial (leucotoxic) or wholesale depressant action on the bone marrow, and in the same way they are caused by the use of such chemicals in industrial work as benzene, trinitrotoluene, nitrobenzene, dinitrobenzene, dinitrophenol *et cetera*. Very interesting is the similar effect of the important antibiotic "Chloromycetin", though it produces aplastic anemia only in a minute fraction of cases (Claudon and Holbrook, 1952). Fatal aplastic anemia due to streptomycin has been recorded also (Womack and Reiner, 1951). The effect is very likely allergic in origin.

As a consequence of modern treatment with hormones, I have here to mention some new syndromes. Such is the syndrome resembling Cushing's disease seen in patients after prolonged use of cortisone (moon face, increase in weight). Important is the osteoporosis with crush fractures of the vertebrae similarly caused by prolonged use of cortisone. Very interesting is the gynecomastia with tense and painful breasts in patients with prostatic cancer treated with high doses of oestrogen, and conversely the manifestation of virilization in women with metastases of breast cancer treated with large doses of androgen (hirsutism, deepening of the voice, enlargement of the clitoris).

In a recent paper Hutchison (1955) described "some new diseases in paediatrics". These are fibrocystic disease of the pancreas ("mucosis"), infantile renal acidosis, hyperchloraemic acidosis, idiopathic hypercalcaemia, idiopathic hypocalcaemia with osteoporosis, and the Lignor-Fanconi syndrome (cystinosis).

Of great importance are new syndromes caused as sequelae of operations in modern surgical treatment. Such are *ulcus jejuni*, the "dumping" (post-gastrectomy) syndrome, the post-cholecystectomy syndrome, and the frontal lobe syndrome, which develops in varying degree after prefrontal leucotomy.

There is a host of new diseases among the industrial diseases: berylliosis (dermatitis, acute and chronic chemical pneumonia—some authors term it pulmonary granulomatosis), fluorosis (joint stiffness and exostoses, calcification of ligaments), asbestosis (diffuse fibrosis of the lungs), graphite pneumokoniosis, bauxite fibrosis of the lungs, lung cancers among workers with chromium, arsenic, nickel *et cetera*. Chronic carbon disulphide poisoning also occurs in viscose rayon factories (Vigliani, 1954) with symptoms of chronic encephalopathy. A very important disease is osteogenic sarcoma in girls employed in painting the dials of luminous watches.

It is not likely that new bacterial diseases will emerge in the future, but it may happen. For virus diseases the probability is much greater. "It is only within the virus diseases that Nature is likely to engender any novelty in infectious disease" (Burnet, 1953). Burnet thinks that there are possibilities of such an occurrence. They may be caused by mutation of so far non-pathogenic viruses or

in the way that "benign infections of birds or animals may find new opportunities of transfer to man" (Burnet, 1953).

The latest new disease, and potentially one of the most important maladies of the future, is radiation disease produced by radioactive fission products. The symptoms are progressive epilation, nausea, vomiting, bloody stools, ulceration of the skin, emaciation, and very severe, sometimes fatal, aplastic anemia. Fortunately so far very few people have succumbed to the disease, but what the future holds is speculative. I will not even touch here on the possibly immense number of victims of the disease in the event of such a disaster as a global thermonuclear war. But nobody at the present time knows the truth of what will be the situation when gigantic atomic plants are working all over the world, replacing the use of coal and oil. It is a problem of effective and safe disposal of the radioactive waste. Experts feel sure that the problem can be solved safely, and that neither the water in the oceans nor the atmosphere will be polluted. Today the task appears to be not so formidable as it was at first thought. We are in complete ignorance at the present time of what would be the effect if, despite all prophylactic measures, such fission products raised the earth's level of radioactivity. Of all the forces coming from outside the organism, radioactivity is the most apt to produce rich mutations of the genes. It is certain that even a slight increase in the radioactivity of the atmosphere would raise the mutation rate of all earth's creatures, from plants to man. From our point of view that might mean an immense variety of new hereditary diseases. According to the geneticists, the full effect of a slight but significant increase of such mutations may not be felt for centuries.

#### Summary.

Diseases and their pattern are changing constantly and progressively. The process is so far the most spectacular in the realm of the infectious diseases. It is spontaneous in some cases, but is mostly due to lessening of poverty, to improved preventive measures, to chemotherapy and to the use of antibiotics. The pattern of some of these diseases has changed to a very great extent (scarlet fever, diphtheria, rheumatic fever, pneumococcal pneumonia, meningococcal meningitis, typhoid fever, erysipelas, syphilis, subacute endocarditis); in other diseases the changing of the pattern is not so conspicuous (for example, tuberculosis). The pattern of hereditary diseases has not changed during the last half-century.

It occurs only exceptionally that a well-defined disease disappears completely all over the world. That has happened with chlorosis, with *prurigo ferox* Hebra and with some virus diseases.

New diseases have appeared and are appearing steadily in considerable numbers. This is due to the tremendously increased consumption of new drugs, especially the antibiotics, and to the hazard of newly installed industrial works. Potentially the most important and most menacing new disease of the future is radiation disease caused by radioactive substances.

I would close my paper with the words of Colin (1946):

If we can judge the future by the present and the past, it seems safe to predict that as long as our planet continues to be inhabitable, change will continue to be a universal law of life.

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## SURGICAL INJURY AND PERIPHERAL CIRCULATORY FAILURE.

By JAN J. SAAVE, M.D.,

Rabaul, Territory of Papua and New Guinea.

ANY major surgical procedure is accompanied by more or less evident disturbances in the peripheral vascular bed. These are complex in nature and are associated with changes of the circulating blood volume. It would appear that the adrenocortical products released under surgical stress are of critical significance, although it is not known to what extent they can modify the peripheral vascular collapse (Frank et alii, 1950). Fegerl (1952) points out that the histamine-like substances released during the immediate post-operative period are also of importance and can cause severe vascular disturbances on an "allergic-hyperergic" basis.

### Pathological Physiology.

The syndrome of peripheral circulatory failure associated with surgical trauma may include the following symptoms and signs: (i) a sense of well-being, (ii) cyanosis of mucous membranes, (iii) a rapid and sometimes almost imperceptible pulse, (iv) a steady fall of blood pressure, (v) lack of evidence of blood or fluid loss during and after operation, (vi) sudden reduction of the circulating blood volume. It requires a great deal of accurate observation not to be deceived by a calm and confident appearance of the patient. He does not arouse any suspicions of an imminent catastrophe. In the cases quoted here cyanosis was actually the only sign observed which suggested an abnormality in the patient's vasomotor control. It may be postulated that the vasostasis, the reduction of the total circulating blood volume and the diminished cardiac output are all initiated by a generalized primary increase in vascular capacity. Other corroborating factors in this type of circulatory failure are the following: (a) escape of plasma from the circulation in the surgically traumatized areas (Fine, 1949; Courtice, 1954); (b) hypoxia of the liver (Shorr, 1951) and the accumulation of vasodepressor material in the blood-stream, with consequent failure in the tone of the peripheral vascular bed (Shorr, 1945; Child, 1952).

Sodium deficiency as a conditioning factor could be disregarded, because of ample pre-operative sodium intake and the absence of gastro-intestinal fluid loss (vomiting, diarrhoea) or undue perspiration during the operation. However, it is known that a severe sodium deficiency can induce peripheral circulatory collapse (Elkinton et alii, 1946). Clinical investigations reveal that the systolic blood pressure will remain the only reliable and accurate criterion indicating the condition of the patient. In addition, the history, the average pulse pressure (Beecher, 1947) and various clinical features (as pulse rate, thirst, vomiting and temperature) have to be considered (Eliermann, 1952). Grant and Reese (1951) have postulated that a systolic blood pressure below 100 millimetres of mercury indicates that the blood volume is about 70% of normal.

The pathological physiology of this type of shock must determine the therapeutic measures to be taken.

It is pointed out by Moore (1951) that the whole blood must be regarded as the best "blood volume expander". It is a carrier of oxygen and carbon dioxide, and provides red cells with their significant "space-occupying" function.

It is stressed by Mayer and Ruben (1951) that in some cases blood transfusion alone failed to raise the arterial blood pressure. However, they achieved very encouraging results with intravenous administration of 1-norepinephrine.

Favourable reports are also available from Moser and Wurnig (1952) and from Miller et alii (1953). However, Sokoloff et alii (1954) insist that the therapeutic efficacy of this drug should be regarded as a prognostic test (pressor response) and an index of severity of shock.

There is no doubt that the administration of 1-norepinephrine is justified and will prove useful in the following conditions: (a) in cardiogenic shock, because of the increased coronary flow and higher oxygen tension of the myocardium (Boyer, 1944; Sayen *et alii*, 1952; Sokoloff *et alii*, 1954); (b) in primary neurogenic shock, because of its vasoconstrictor effect; (c) in the decompensated phase of shock, because of increased blood supply to the brain and heart, and to counteract the vasodepressor action of the accumulated ferritin (Shorr *et alii*, 1950). It could be anticipated that the combined use of 1-norepinephrine and whole blood would prove very efficacious in cases of the late decompensated phase of shock.

However, the administration of 1-norepinephrine is strictly contraindicated in the early compensated phase of shock, because of its strong vasoconstrictor effects.

The pharmacology of this drug may be summarized as follows: (i) It is a sympathomimetic (Euler, 1946, 1951), and probably related to "Sympathin 'E'" (Canon and Rosenbluth, 1933). (ii) It is a vasoconstrictor, and increases total peripheral resistance and systolic and diastolic blood pressure. (iii) It increases cerebral blood flow (King *et alii*, 1952; Sokoloff *et alii*, 1954) and coronary blood flow in hypotensive patients, and decreases splanchnic, skin and renal blood flow. (iv) It is not a metabolic stimulant (Goldenberg, 1951). (v) When administered intravenously it acts immediately, and it is also rapidly inactivated in the body (Euler, 1951). Untoward reactions are few—namely, headache, sweating, diarrhoea and tissue necrosis, due to paravenous administration (Bergmann, 1952).

#### Reports of Cases.

The following is a description of three cases of the syndrome of peripheral circulatory failure (so-called surgical shock).

**CASE I.**—A male New Guinea native, aged approximately twenty years, was admitted to hospital on February 10, 1953, with acute abdominal pain in the left hypochondrium. On being questioned, the patient admitted to having had an attack of severe pain below the left costal margin three days prior to his admission to hospital. He also complained of distressing diarrhoea. When he was examined, his temperature was 101° F. The pulse rate was 120 per minute, his breathing was predominantly thoracic and his respiration rate was 32 per minute. The blood pressure was 115 millimetres of mercury, systolic, and 75 millimetres, diastolic. Auscultation revealed râles and rhonchi at the base of the left lung and impairment of the percussion note. The left lower intercostal spaces were acutely tender on palpation. Examination of the abdomen revealed an enlarged spleen, tender to touch and occupying the entire left hypochondrium. There was also an ill-defined mass in the epigastrium. The left upper and lower abdominal reflexes could not be elicited. Rectal examination revealed no abnormalities. A blood count gave the following information: The haemoglobin value was 92%, the erythrocytes numbered 4,700,000 per cubic millimetre, and the leucocytes numbered 12,000 per cubic millimetre, 69% being neutrophilic cells, 2% monocytes and 29% lymphocytes. The blood clotting time was greatly increased. There were no malarial parasites in the blood smear. The diagnosis could not be decided upon at this stage, and the patient was admitted to hospital for further observation. An acute abdominal condition and early lobar pneumonia were considered in the differential diagnosis. The patient was given 300,000 units of procaine penicillin (aluminium stearate) once a day, 500,000 units of crystalline penicillin every six hours, 10 grains of quinine as a malaria suppressive twice a day, and only fluids by mouth.

Within the next forty-eight hours his condition did not improve. His temperature was up, his tongue was furred and there was rigidity of the abdominal wall. The diagnosis of an acute abdominal emergency was made, and the patient was operated upon under general anaesthesia on February 12. The abdomen was explored through a left paramedian incision. The peritoneal cavity was free of blood, but contained a small amount of cloudy fluid. No abnormalities of the viscera were found except in the spleen. This organ was enlarged and contained multiple wedge-shaped infarcts. Their appearance varied; some of them were red and distinctly elevated, the others were of a yellow-grey colour. Since the main arterial trunk was occluded, splenectomy was performed. The blood loss at operation was negligible. On February 13 at 9 a.m. the patient appeared calm and in

apparently good condition. At 1 p.m. he was fully conscious, his extremities were very cold and his pulse rate was 160 per minute. His blood pressure could not be registered on the sphygmomanometer. No heart sounds were heard on auscultation. The mucous membrane of the mouth appeared cyanotic. Intramuscular injections of "Coramine" and caffeine-sodium benzoate were of no avail. An intravenous slow drip administration (20 drops per minute) of 1-norepinephrine in normal saline (12 milligrammes per litre) was commenced at 1.45 p.m. The blood pressure was recorded every fifteen minutes. At 3 p.m. it was 105 millimetres of mercury, systolic, and 65 millimetres, diastolic. Unintentional withdrawal of the 1-norepinephrine infusion resulted in a slow but steady fall in blood pressure. Administration of 50 milligrammes of cortisone produced no improvement. The general condition of the patient began to deteriorate, and he succumbed at 5.45 p.m. before a blood transfusion could be organized.

Necropsy revealed congestion of the bases of both lungs, vegetations on the mitral valve and much dilated blood vessels in the gastro-intestinal tract.

**CASE II.**—A male New Guinea native, aged approximately twenty-six years, was admitted to the hospital on June 16, 1953, with a strangulated indirect inguinal hernia on the right side, of ten hours' duration. On examination of the patient, his temperature was 99° F., his pulse rate was 100 per minute, and his blood pressure was 130 millimetres of mercury, systolic, and 95 millimetres, diastolic. Auscultation revealed no abnormalities of the lungs. The abdomen was moderately rigid and tender on palpation and percussion.

The patient was operated on under general anaesthesia on the same day. A parainguinal incision was used and a sliding hernia was disclosed. The length of the incarcerated loop of the small gut was 20 centimetres (eight inches). The peritoneal sheen was lost, the bowel was distended and it was almost Burgundy-red in colour. Immobilization and freeing of the intestinal loop from the hernial sac took a considerable amount of time, because of obliteration of the neck by thick layers of omentum. It was adherent to the base and along the walls. Ten centimetres (four inches) had to be resected. The bowel appeared viable and was returned to the peritoneal cavity. The blood loss during the operation was not more than 50 cubic centimetres. The immediate post-operative recovery was uneventful.

However, thirty hours after operation the patient became restless. His pulse rate was 140 per minute; his blood pressure was 90 millimetres of mercury, systolic, and 55 millimetres, diastolic. His temperature was 101° F. and he was perspiring profusely. He also complained of generalized abdominal pain. Blood examination revealed no malarial parasites. One hour later the blood pressure was even lower—75 millimetres of mercury, systolic, and 40 millimetres, diastolic. The mucous membranes of the mouth were conspicuously cyanotic.

A tentative diagnosis of post-operative peritonitis accompanied by peripheral circulatory failure was made. An intravenous infusion of 1-norepinephrine in normal saline (12 milligrammes per litre) was started immediately. The improvement was obvious, and it was possible to keep the systolic blood pressure at approximately 100 millimetres of mercury. As soon as the administration was stopped the patient's condition began to deteriorate, and there was a fall of blood pressure to 85 millimetres of mercury, systolic, and 45 millimetres, diastolic. It was decided at this stage to give him a transfusion of 500 cubic centimetres of whole blood. Recovery was indeed amazing; his blood pressure stayed at 105 millimetres of mercury, systolic, and 75 millimetres, diastolic, his pulse rate was 90 per minute, and he was sitting up. Sixty hours after operation his abdomen was soft on palpation; his temperature was 98.4° F. He complained only of frequent watery motions and frequent micturition. The patient was discharged from the hospital as cured on July 17.

**CASE III.**—A female New Guinea native, aged approximately forty years, was admitted to the hospital with a diagnosis of prolapse of the uterus. Preliminary examination revealed no other abnormalities, and the patient was submitted to Gilliam's suspension operation under spinal anaesthesia. Her pre-operative blood pressure was 120 millimetres of mercury, systolic, and 85 millimetres, diastolic. Her pulse rate was 80 per minute. She received the usual pre-operative anti-malaria treatment (quinine dihydrochloride, 10 grains twice a day). The suspension of the uterus was completed without any mishaps. The amount of blood lost during the operation was not more than 30 cubic centimetres. Forty-eight hours after operation, peripheral circulatory failure developed. The mucous membranes of the mouth were cyanotic, the blood pressure could not be registered on the sphygmomanometer, and her pulse was imperceptible. How-



ever, she was fully conscious and astonishingly "well". An intravenous slow drip administration of eight milligrammes of 1-norepinephrine in one litre of normal saline produced only transient but definite improvement. The systolic blood pressure was just under 100 millimetres of mercury. The patient received also six litres of oxygen per minute. The intravenous infusion was stopped now and then and the blood pressure was recorded every five minutes. It was evident that the withdrawal caused a noticeable and progressive fall of blood pressure. It was decided to give her a transfusion of 500 cubic centimetres of whole blood simultaneously. The drip administration of 1-norepinephrine was re-started, the systolic blood pressure was kept at about 100 millimetres of mercury and the blood transfusion was commenced. Thereafter the recovery of the patient was undisturbed.

#### Summary.

The pathophysiology, clinical characteristics and treatment with 1-norepinephrine of peripheral circulatory failure are discussed.

Three cases are reported to illustrate the occurrence of this syndrome, presumably as a result of surgical injury.

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#### Reviews.

**Morbidity in the Municipal Hospitals of the City of New York: Report of an Exploratory Study in Hospital Morbidity Reporting.** By Marta Fraenkel, M.D., and Carl L. Erhardt; 1955. New York: Russell Sage Foundation. 9" x 6", pp. 230, with 56 tables. Price: \$4.50.

In order to provide a basis for administration and planning for the future in the hospitals of a large city, the New York Board of Health, supported by the Russell Sage Foundation, carried out a comprehensive in-patient survey of the 31 New York municipal hospitals over a six-months period. In this report there are 56 tables covering all aspects of the matter, with explanatory text. In particular, the survey shows that the need for hospitalization of children has been shrinking over the years, while nearly one-sixth of all patients discharged from hospital during the survey period were aged sixty-five years or over. While the data offered are specifically related to conditions in New York, many of them have a general application and could be usefully availed of by hospital planners everywhere.

**Australia in the War of 1939-1945: Series Four, Civil; Volume 3. War Economy, 1939-1942.** By S. J. Butlin; 1955. Canberra: Australian War Memorial. Obtainable at all booksellers. 9½" x 6½", pp. 534, with 21 illustrations. Price: 25s.

THIS is one of the series dealing with Australia in the war of 1939-1945; it relates to the economic organization in Australia during the first two years of the war. There is first a general discussion of Australian economic policy, such as it was, in relation to defence during the pre-war years. After the Imperial Conference of 1937, at which the need for and scope of preparations to meet the deteriorating international situation were indicated, there came a stage of activity in the fields of munitions, aircraft, wartime exports, war supplies and manpower, but there was practically no effective economic planning. Once war broke out, the first matters to be organized were foreign exchange transactions and prices control, the latter having been regarded as essential to prevent profiteering. The author then deals with the organization and protection of those industries on whose exports Australia traditionally depends for overseas credits, and on the moulding of these industries to meet the needs of the wartime domestic economy. Bound up with this was the question of imports, and what among them was essential or unessential. Shipping, shipbuilding, and the Shipping Control Board are then dealt with.

All aspects of wartime economy are thereafter described, from budgets, supply and production, to transport, lend-lease and manpower, leading up to a survey of the economic situation on the eve of Pearl Harbour. Although this volume is written primarily from the point of view of the economist, it is never too technical or abstruse, and it sets out the various elements of the story lucidly and interestingly for the average reader with a wish for knowledge on this side of the war effort.

**Ciba Foundation Symposium on Experimental Tuberculosis Bacillus and Host, with an Addendum on Leprosy.** Editors, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Cecilia M. O'Connor, B.Sc.; 1955. London: J. and A. Churchill, Limited. 8" x 5½", pp. 408, with many illustrations. Price: 42s.

THIS is a Ciba Foundation symposium consisting of 23 articles on tuberculosis and four on leprosy. These articles deal only with the nature, constitution and properties of the bacilli concerned and the reactions of the host tissues, and have nothing to do with clinical or therapeutic aspects

of the disease. Thus the opening articles deal exhaustively with the proteins, mycolic acids, mycobactin and polysaccharides of the tubercle bacillus; then tissue reactions in general and in specific response to the various stimuli inflicted by the bacilli and their products are dealt with. Thereafter immunity and sensitization are discussed, and tissue reactions as influenced by chemotherapy, and drug resistance. In the addendum on leprosy, its pathogenic relationship to tuberculosis is discussed, together with the tissue responses to *Mycobacterium leprae*, and the immunological and physiological basis of reactions.

As might be expected, much of the matter is highly technical, but it is very well arranged and presented, and the discussions which follow each article are helpful, and shed much extra light on them. Notwithstanding the big advances which have been made in treatment, both tuberculosis and leprosy are still widespread and important diseases, and their problems have by no means been solved, so that the assembling, in one volume such as this, of so many basic data is a useful and timely service which should be much appreciated by workers on these diseases everywhere. Collaterally, much of the information offered is not restricted to the Mycobacteria, but applies to the action of infecting organisms in general, so that the book actually has a wider field of interest than its title suggests.

**The Zoonoses in their Relation to Rural Health.** By Karl F. Meyer; 1955. California: University of California Press. 9½" x 6", pp. 54, with nine illustrations. Price: \$1.00.

This booklet is written more particularly for those countries whose public health and agricultural services, if they exist at all, are poorly developed and organized. Nevertheless, it contains much that, properly applied, can benefit the better-organized countries too. The diseases of animals which can affect man are of major importance everywhere. They include some of the earth's major diseases; brucellosis, for instance, is world-wide in one or other of its forms, and attacks millions of people each year; apart from the loss of efficiency it causes among its human victims, the annual losses in animals run into many millions of pounds.

Karl Meyer points out that notification of animal disease and the collection of figures on its occurrence and effects are as important as the notification of infectious diseases among the human population, and that the control of vectors and reservoirs of zoonoses is at least as essential as proper waste disposal and the provision of safe water supplies and sanitation. He then discusses the major zoonoses: anthrax, brucellosis, encephalitis, hydatid disease, leptospirosis, plague, Q fever, rabies, salmonellosis, trichinosis, bovine tuberculosis and tularemia. Of these, only plague, rabies and tularemia do not occur in Australia; only Q fever, rabies and tularemia are not officially notifiable here. The omission of Q fever from our list is interesting; certainly more cases of Q fever than anthrax, bilharziasis, trichinosis and many other notifiable diseases occur. Meyer points out that universally the dairy and livestock industries show little interest in the control of Q fever, simply because the infected animals are not visibly affected. As for man, the only feasible protection, in the absence of any attack on the reservoir or vectors, would be prophylactic vaccination of the exposed population.

Meyer then mentions those zoonoses of minor and local importance, of which ectoparasitic and fungous infections, psittacosis and teniasis are of interest in Australia.

This booklet is well written and produced, even if it does startle us with its description of animal "lice (*sarcoptes* sp.)" which may cause "human scabies", and its illustration of bananas growing on a fair representation of a coconut palm. However, these are minor errors, and apparently the only ones, and do not detract from the very real value of the publication.

**Animal Agents and Vectors of Human Disease.** By Ernest Carroll Faust, M.A., Ph.D.; 1955. Philadelphia: Lea and Febiger; Sydney: Angus and Robertson, Limited. 9½" x 6½", pp. 660, with 225 illustrations, one in colour. Price: £5 5s.

THERE has been no book on this subject published in recent years to compare with Professor Ernest C. Faust's "Animal Agents and Vectors of Human Disease". For completeness, systematization and ease of reference it is unsurpassed, and while the date on the title page is 1955, it contains many major references dated 1953, which is evidence of its being as up to date as is possible. The first section on general information and orientation contains many useful tables, including a complete systematic classification of animal agents which places them all in their correct perspective, as regards both their zoological status and their relationship to disease. Each chapter deals thoroughly with one

group of agents, their characteristics, their history, nomenclature and distribution, and their association with disease; the disease is then dealt with in all aspects; treatment is fully and fairly assessed and control is discussed. There follows, for each chapter, an adequate summary, and then a comprehensive but not indiscriminate bibliography. The illustrations are numerous and worth while. The indices are adequate. The book is excellently produced, and there are very few misprints. "Animal Agents and Vectors of Human Disease" can be strongly recommended to all, from students to specialists.

**Clinical Neurosurgery: Proceedings of the Congress of Neurological Surgeons, New Orleans, La.** Editor-in-Chief, Raymond K. Thompson, M.D.; 1955. Baltimore: The Williams and Wilkins Company; Sydney: Angus and Robertson, Limited. 9" x 6½", pp. 210, with 89 illustrations. Price: 86s.

THIS book records three papers presented by Sir Geoffrey Jefferson on "Changing Views on the Integration of the Brain", trigeminal neurinomas and compression of the optic pathways by intracranial aneurysms, and panel discussions on the anatomy and physiology of the frontal lobe, psychosurgery, and the use of fluids and electrolytes in the management of the neurosurgical patient. These are, in effect, a record of the proceedings of the Congress of Neurological Surgeons held at New Orleans during 1954, and represent the latest opinions of many distinguished workers. Each of the subjects has a highly specialized appeal, but appears to have been covered very thoroughly, and to offer valuable contributions. Production is excellent.

**Sexual Offenders.** By Norwood East, M.D., F.R.C.P.; with extracts from the "Psychological Treatment of Crime", by East and Hubert; 1955. London: Dellsie, Limited; Sydney: Father and Son Welfare Movement of Australia (distributors). 7½" x 5", pp. 102. Price: 15s. 6d.

MOST aspects of forensic medicine present a conflict between theory and practice, between medical and legal interpretations, which is very hard to reconcile. Particularly is this so in the matter of sexual offenders. In the booklet of this title, by Sir Norwood East, there is evidence of conflict in the legal list of sexual offences on the one hand, and the attempt to define sexual perversions on the other. Sexual offenders are those who engage in certain illegal sexual activities: broadly, unnatural offences, rape, indecent assaults and exposure, defilement of girls under sixteen, incest, procuration, abduction, bigamy and prostitution. Sexual perversion is defined in this book as sexual activity in which complete satisfaction is obtained without the necessity of heterosexual intercourse; it must be persistently indulged in, in reality or fantasy, and not merely as a substitute for a preferred heterosexual activity which is unobtainable for some reason. It will be seen that the legal definitions are precise, while this medical definition is vague, full of loopholes, and incomplete. The essential issue is here regarded, not as the apparent abnormality or perversity of the sexual manifestation, but its dominance, and the sexual pervert is the individual who shows an undue persistence or dominance of what, "for the majority, would at most constitute a minor sexual component or urge". Thus an assault on an unwilling woman or a young girl, it is specifically stated, is not necessarily evidence of mental or sexual abnormality, any more than is occasional indulgence in homosexuality as an "adventure" or a "temporary convenience". This more-than-broad-minded approach to the subject nullifies any possible reliance on the measures advocated for the treatment of sexual offenders by psychoanalysis in the latter part of the booklet.

**Advances in Internal Medicine.** Edited by William Dock, M.D., and I. Snapper, M.D.; Volume VII; 1955. Chicago: The Year Book Publishers, Incorporated. 9" x 6", pp. 312, with many illustrations. Price: \$8.50.

THE seventh volume, for 1955, of "Advances in Internal Medicine" contains eight sections by various authors. Like all compilations of this type, its articles consist mainly of reviews of recent literature and assessments of the latest knowledge and opinions on the various subjects; in this instance this has been fairly well carried out. The position with regard to the association of renal lithiasis with the collagen diseases and the place of hyaluronidase, infection and endocrine and metabolic defects is put very well. The section on hepatic coma is good enough as far as it goes, but establishes only that no definite information of any value exists, and the same applies to pancreatitis. "The Clinical Significance of Serum Mucoproteins" is much ado about very little, as the measurement of these gives prac-



tically no information, and that of doubtful specificity. Disorders of the adrenal cortex are well dealt with, as are diseases of the pericardium and the nephrotic syndrome. The clinical usefulness of pulmonary function tests is not convincingly demonstrated. Nevertheless, as a faithful record of recent work, the book fulfils its function—it is in the selection of its subjects that it is unfortunate.

**History of the Second World War: United Kingdom Medical Series.** Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.A., M.D., F.R.C.P., F.R.C.S. "The Civilian Health and Medical Services", edited by Arthur Salusbury MacNalty, K.C.B., M.A., M.D., F.R.C.P., F.R.C.S.; 1955. Volume II: The Colonies, The Medical Services of the Ministry of Pensions, Public Health in Scotland, Public Health in Northern Ireland. London: Her Majesty's Stationery Office. 9½" x 6½", pp. 418. Price: 45s.

TOTALITARIAN war brings to the civilian health and medical services not only new problems, but work of a magnitude and an urgency never met with on such a large scale or so prolonged in peace time. In Volume II of "Civilian Health and Medical Services", edited by Sir Arthur S. MacNalty, the way in which the civilian services carried out their work is amply demonstrated. Part I concerns the Colonies, Part II the work of the Ministry of Pensions, Part III Scotland, and Part IV Northern Ireland. The first chapter describes colonial medical services as they existed prior to the outbreak of war. Then the problems of Malta, under the intensive bombing it endured, are dealt with. With the most crowded population in the world, and a limited area, these problems were acute. Rationing had to be severe, with a noticeable effect on the health of the population; its one good effect was the virtual disappearance of Malta fever because of slaughtering of goats for food and governmental control of pasteurization. The epic of the Maltese people's steadfastness is well known.

Hong Kong's problems were different, since it was assaulted and captured. During the preliminary bombing and the fighting all services broke down; after the surrender there was chaos, and only the fact that the Director of Medical Services was permitted to carry on to a limited extent provided some mitigation of the disorganization. Deliberate and systematic starvation by the Japanese, with the avowed intention of reducing the population of one and a half millions to one-third, fully succeeded in achieving this object. The medical services, with limited supplies and improvised equipment, did an excellent job of caring for all they could reach—but nevertheless were accused of collaboration with the Japanese.

Malaya, Singapore and North Borneo were in the same situation as was Hong Kong. During 1942 all medical control deteriorated, as the Japanese provided little towards it, and by 1943 semi-starvation had set in. Malaria, always only kept in check by intensive control measures, broke out in disastrous epidemics, with no drugs for treatment. Dysentery and deficiency diseases were rife.

British Somaliland was occupied by the Italians, and their treatment of the inhabitants was civilized. Palestine, Cyprus, Gibraltar and Ceylon carried on more or less normally under their increased burdens.

The Ministry of Pensions has, in addition to its civilian responsibilities, service responsibilities equivalent to those of the Australian Repatriation Department, and its methods of meeting its responsibilities are the same for civilians as for servicemen. It was concerned with compensation for, treatment of, and a certain amount of rehabilitation after, injuries suffered by civilians as a direct consequence of enemy action. The records for Scotland and Northern Ireland, as presented, are routine and uninteresting.

This volume provides a complete record of everything within the scope of its title. Production is excellent.

**The Abnormal Pneumoencephalogram.** By Leo M. Davidoff, M.D., and Bernard S. Epstein, M.D.; Second Edition; 1955. Philadelphia: Lea and Febiger; Sydney: Angus and Robertson, Limited. 10" x 7½", pp. 518, with 696 illustrations. Price: £8 1s. 3d.

THE first impression of this book is that too much has been made of the subject and that the enthusiasm of the authors for this investigation has led them to write more about it than is of practical usefulness. On reading the book carefully, however, this view is altered. The conclusion is that thorough attention to the subject has enabled the authors to reveal to the practical neurosurgeon how much information he can derive by means of pneumoencephalography over and above that which ordinary use of this investigation provides.

In their preface to the second edition the authors recognize the popularity of angiography and other special methods in investigating neurological cases, which have in many instances supplanted pneumoencephalography and in fact excelled it, and as they state pneumoencephalography is not performed quite as often as previously.

It is interesting to see the views which they have published in which the pneumoencephalogram discloses small aneurysms in the circle of Willis, and other lesions which are better displayed by angiogram, none the less revealed by this method. This provides the reminder that one should examine the small details of the shadows in the pneumoencephalogram and seek to explain the nature of every one of them.

It is of great interest to see the application of "lamina-grams" to pneumoencephalograms, and there is one striking illustration of a mid-line laminogram taken in the lateral view of a pneumoencephalogram and providing a very clear picture of the outline and position of the third ventricle, cerebral aqueduct and fourth ventricle, which in the picture are actually quite faintly filled with air.

The authors' attention to the pneumoencephalographic appearances in vascular abnormalities within the head is of interest.

In any neurosurgical department, quite a large number of people have pneumoencephalograms done because they have had epileptic phenomena, often with nebulous suggestions of focal features and because there is no reliable witness of the form of the fits which have occurred. The electroencephalogram is quite often inconclusive, and this can occur in the presence of an uncalcified angiomatous malformation on or in the brain. Study of such a book as this could result in prompt recognition of a patient's condition and so in earlier treatment.

Of special interest is the full detail with which the authors discuss the many lesions which can lead to atrophic changes in the pneumoencephalogram, and this is something which is of daily concern to the neurosurgeon.

It would be profitable for any neurologist, especially a neurosurgeon, to peruse this book, and in any hospital which has had the good fortune to be able to appoint a neuro-radiologist or a neuroradiographer, a copy of this book should be at hand and carefully studied. In other words, the book is very thorough and is undoubtedly a classic in that it provides a full statement of the uses and value of this form of investigation in 1955.

**Practitioners' Conferences: Held at The New York Hospital—Cornell Medical Center.** Edited by Claude E. Forkner, M.D., F.A.C.P.; 1955. New York: Appleton-Century-Crofts, Incorporated. 8½" x 6", pp. 426, with many illustrations.

THIS is a record of discussions, on a variety of subjects, by New York practitioners and a panel of experts assembled at the New York Hospital-Cornell Medical Center. The discussions were informal, and all present were encouraged to join in. Subjects ranged through influenza and primary atypical pneumonia, the harmful effects of tobacco, coronary thrombosis, acute and chronic sinusitis, the present status of ACTH and cortisone, headache, and disorders of the feet, with many others. Much of the matter was in the form of clinical meetings. Frequently records of this nature make very dull reading, but those in this volume have been well selected and reported, and the actual discussions are full of meat, while the summaries by the chairman represent concise statements of the latest developments. The book gives indisputable proof of the value of such conferences. It is well produced, and no misprints were noticed.

**Surgery of the Small and Large Intestine.** By Charles W. Mayo, M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 8½" x 6", pp. 340, with 94 illustrations. Price: \$9.00.

THIS book from the Mayo Clinic sets out to present concise descriptions and illustrations of the more common surgical procedures performed for lesions of the small and large intestine, and it certainly succeeds in its object. The main steps of the various operations are clearly described both in the drawings and in the text. The freedom from typographical errors is worthy of note; but on page 290 we wonder whether the failure to mention a row of interrupted cotton stitches in the posterior serosal surface is intentional. With surgery of the small and large intestine there are not many wide differences of opinion at present as to the best type of procedure to adopt, and thus, although only a single

technique is described here for each operation, this suffices. However, an important omission is any reference to mucosal grafted ileostomy as described by Turnbull. On the other hand, while there is accord as to the general principles of operations on the small and large bowel, there are still controversial points in minor details of operative technique and in the pre-operative and post-operative management of these cases. For instance, it is not widely accepted that "prophylactic Dicumarol therapy is worthy of consideration in those patients who have a history of previous post-operative thrombophlebitis or pulmonary embolism". Also, it is not usually agreed that "a small portion of the gluteal muscle" should be removed during an abdomino-perineal resection of the rectum for cancer or that an extraperitoneal closure of a colostomy need be carried out in two stages. Throughout the book the teachings are predominantly American, even to the extent of calling Schmieden's stitch the baseball stitch, but in the final chapter the anatomy and description of anal fistulae follow that of Saint Mark's Hospital, London. This book may be strongly recommended to all those interested in surgery of the small and large bowel, but its chief place will probably be in the instruction of post-graduate students.

**Mysterious Waters to Guard: Essays and Addresses on Anaesthesia.** By Wesley Bourne; 1955. Oxford: Blackwell Scientific Publications. 9½" x 6", pp. 418. Price: 42s.

THE highly erudite character of Wesley Bourne's book, "Mysterious Waters to Guard", is a credit to his catholicity of interest and to his learning. Indeed, a better title, perhaps, would have been "The Philosophical Basis of Anaesthetics".

Subsidized by a grant from the United States of America, written by a Canadian, published in England and printed in Holland, the book truly possesses an international character. It comprises a selection of the articles and addresses delivered by the author over a period of nearly thirty years, and so illustrates the development of his thoughts, literary skill and practical activities in that time. The title refers to the intimate watery medium in which the cells of the human body live, and to the imperative necessity to safeguard their welfare in the face of the inimical possibilities associated with the induction of narcosis and anaesthesia. Unfortunately, Dr. Bourne is an inveterate collector of strange and abstruse terms, which he is apt to employ with considerable abandon, and sometimes evident inaccuracy. The not infrequent use of frank neologisms suggests that he must be an earnest disciple of the late Noah Webster.

Opening with a brief foreword by Cornelle Heymans, Professor of Pharmacology of the University of Ghent, Belgium, and Nobel Prizeman in 1938, there follows an erudite and learned preface by the author. These are succeeded by 32 essays, which exhibit a progressive improvement in style and maturity, and then a very fine brief epilogue. Towards the end there is a symposium, "The Role of Basic Sciences in Anaesthesia", to which the author and the Professors of Pharmacology, Anatomy, Biochemistry and Physiology of the McGill University, Montreal, make contributions. The lucidity of these is in refreshing contrast with the author's usual style.

It is a great pity that Dr. Bourne, instead of quoting his several articles *verbatim*, did not use them as a basis for a really good work on the fascinating subject he has chosen.

There is no question whatever about Dr. Bourne's great scholarship, nor of the enviable position he occupies as a man of learning and science. As a writer, however, he often lacks those essential virtues of simplicity, brevity and clarity. His turgidity of style and the tendency to obscurity in presentation, together with the intolerably repetitive character of many of the articles, make the book so tedious to read as to discourage its complete perusal. Nevertheless, the latter part of his work contains much of value, especially in regard to the organization of teaching in anaesthetics.

The book contains 45 fine photographs of various anaesthetists and others interested in either anaesthetics or its related subjects, including three prominent Australians. A few typographical errors occur, those in the first line of page 146, covering seven consecutive words, being outstanding. There is an excellent bibliography, comprising 621 references, and an exhaustive index covering the final 71 pages. A second edition, entirely recast and free of prolixities, affectations and errors, would indeed be a valuable addition to medical literature.

## Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"World Distribution of Spirochetal Diseases: 3. Leptospirosis: Basic Sources", by the Department of Medical Geography, American Geographical Society; 1955. New York: American Geographical Society. 38" x 26". Price: \$1.25 folded, \$1.50 flat.

The address of the Society is Broadway, at 156th Street, New York 32, N.Y., U.S.A.

"The Year Book of Medicine (1955-1956 Year Book Series)", edited by Paul B. Beeson, M.D., Carl Muschenheim, M.D., William B. Castle, M.D., Tinsley R. Harrison, M.D., Franz J. Ingelfinger, M.D., and Philip K. Bondy, M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 8" x 5", pp. 712, with 121 illustrations. Price: \$6.00.

One of the Practical Medicine Series of Year Books.

"Fractures of the Facial Skeleton", by N. L. Rowe, F.D.S.R.C.S. (Eng.), L.R.C.P. (Lond.), M.R.C.S. (Eng.), L.M.S.S.A. (Lond.), H.D.D.R.C.S. (Edin.), and H. C. Killey, F.D.S.R.C.S. (Eng.), L.R.C.P. (Lond.), M.R.C.S. (Eng.), L.M.S.S.A. (Lond.), H.D.D.R.C.S. (Edin.), with a Foreword by Sir Reginald Watson-Jones, B.Sc., M.Ch.Orth., F.R.C.S., F.R.A.C.S. (Hon.), F.A.C.S. (Hon.); 1955. Edinburgh and London: E. and S. Livingstone, Limited. 10" x 7", pp. 960, with 1231 illustrations. Price: £6.

Intended for the orthopaedic surgeon, the dental surgeon and the specialist in facio-maxillary surgery.

"A Short Textbook of Surgery", by C. F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S. (Ed.), F.R.F.P.S. (Glas.), Hon. F.A.C.S.; Sixth Edition; 1955. London: J. and A. Churchill, Limited. 9½" x 6", pp. 636, with 227 illustrations. Price: 37s. 6d.

Intended for undergraduate and post-graduate students.

"A Short Textbook of Midwifery", by G. F. Gibberd, M.B., M.S. (Lond.), F.R.C.S. (Eng.), F.R.C.O.G.; Sixth Edition; 1955. London: J. and A. Churchill, Limited. 8½" x 5½", pp. 604, with 199 illustrations. Price: 30s.

The first edition was published in 1938.

"Obstetrical Roentgenology", by Robert Berman, M.D., F.A.C.S.; 1955. Philadelphia: F. A. Davis Company, Publishers. Sydney: Angus and Robertson, Limited. 9½" x 7", pp. 614, with 486 illustrations. Price: £6 17s. 6d.

Based on the experience of approximately 2500 obstetrical cases.

"Practical Section Cutting and Staining", by E. C. Clayden, F.I.M.L.T.; Third Edition; 1955. London: J. and A. Churchill, Limited. 8" x 5½", pp. 160, with 26 illustrations. Price: 12s. 6d.

Divided into three sections dealing with paraffin wax sections, frozen sections and celloidin sections.

"Alfred Hospital Clinical Reports", Volume V; 1955. Melbourne: Published by Alfred Hospital. 10" x 6", pp. 132, with many illustrations.

There is an article on Prince Alfred after whom the hospital is named and this is followed by 14 articles of clinical interest.

"The M.B., B.S. Finals: A Collection of the Papers Set at the London M.B., B.S. Examination for the Years 1946-1955, Classified and Arranged in Suitable Sub-divisions", by Francis Mitchell-Heggs, T.D., M.B., B.S. (Lond.), F.R.C.S. (Eng.), F.R.C.S. (Edin.); Fourth Edition; 1955. London: J. and A. Churchill, Limited. 8" x 5½", pp. 88. Price: 10s. 6d.

The third edition was published in 1947.

"Office Procedures", by Paul Williamson, M.D.; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 11" x 8", pp. 422, with many illustrations. Price: £6 5s.

Deals with minor "office" procedures. He advocates frugality in the equipping of an office and describes "the use of material at hand to do the job".



## The Medical Journal of Australia

SATURDAY, JANUARY 21, 1956.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

### FIFTY YEARS OF ADVANCE IN ANÆSTHETICS.

In this issue there appears on page 77 an address by Dr. Harry J. Daly, who was president of the Section of Anæsthesia at the Ninth Session of the Australasian Medical Congress (British Medical Association) held at Sydney in August last. This address will be read with interest particularly by senior members of the profession, many of whom spent their early days after graduation becoming proficient in the administration of chloroform and in the use of the Clover inhaler. In those days the fear of chloroform had not developed as it has today, and many abdominal sections were carried out under its agency. The anæsthetization of a patient by means of a Clover inhaler was undoubtedly effective, but it was not always a pleasant sight. More often than not a considerable degree of cyanosis was produced, and the patient cannot have found the proceedings much to his liking. Many a time he put up a good fight before he was ready to be wheeled into the operating theatre. The changes that have taken place since those days have been enormous, as those medical practitioners who have had to submit their own bodies to surgical operation in recent years can testify. The anæsthetist has become a highly specialized person, and efficiency in the practice of anæsthesia is not acquired without special study. It is true that there is a difference between the type of anæsthesia required in everyday general practice where procedures are more or less trivial, and that which should be found in centres where the performance of major operations is an everyday occurrence. What Dr. Daly calls his reminiscences could be duplicated by many specialist anæsthetists of his vintage. He shows how one anæsthetic agent after another was introduced, how some of them have been discarded

and others have gained a permanent place in the anæsthetist's armamentarium. The attention of those who read Dr. Daly's address—and they will be numerous—is directed to a larger "collective review" published recently by Henry K. Beecher and Charlotte Ford, of Boston, Massachusetts.<sup>1</sup> Their review is entitled "Anæsthesia: Fifty Years of Progress". It takes the reader into aspects of the subject which Dr. Daly could not reach in the short time at his disposal.

Beecher and Ford begin by stating that anæsthesia forms a bridge between pharmacology and surgery. Anæsthesia not only ushered in the modern era of surgery, but it also marked the beginning of the growth of pharmacology as a science. It is undoubtedly true that the anæsthetist has unusual opportunities for furthering the progress of surgery and for advancing pharmacology. They insist that it has been impossible to prepare a sound review of the last fifty years of anæsthesia without constant reference to earlier years. They quote Clark as having stated in 1938 that only three discoveries of major importance to pharmacology were made in the nineteenth century: anæsthetics, antiseptics and endocrine therapy. They go into the early history of anæsthesia and remark that the demonstration of William T. G. Morton at the Massachusetts General Hospital on October 16, 1846, certainly initiated the anæsthesia age. They set out a list of some fourteen events connected with anæsthesia which occurred between the years 1853 and 1899, and they point out that in these diverse innovations there is a common factor—they were all developments which made possible previously impossible therapeutic procedures (usually operations). This they regard as being the essential nature of an important advance. Before 1905, which is the year marking the beginning of their review, the following had been discovered: nitrous oxide, ether, chloroform, ethyl chloride, ethylene, cyclopropane (but not its anæsthetic powers), cocaine, the esters of aminobenzoic acid, procaine and the barbiturates. We know that these substances were not all used in actual practice until the twentieth century was quite well advanced. Dr. Daly describes the introduction of some of them and their use by well-known practitioners in Sydney, and we know that the recording of a personal incident serves to impress the subject on a reader. Referring to ethylene, which is mentioned at some length by Dr. Daly, Beecher and Ford point out that historically it is of importance because of its speed of action and slight disturbance of the carbohydrate metabolism. In these respects it is an "interesting approach" toward the ideal anæsthetic. Divinyl ether is mentioned. It is, we are told, of historical interest because it is the result of an attempt to relate anæsthetic action to chemical structure. It has been used for major surgical operations; but, "like one of its parents, ethylene, divinyl ether does have a more rapid action than diethyl ether". Whether this is the result of the instability of the compound or for other reasons, it is found to be a troublesome irritant, and to produce liver damage under some circumstances. Cyclopropane was first prepared in 1882; but it was not until 1928 that it was discovered to be a potent anæsthetic agent. Beecher and Ford find that an important characteristic of the drug, which has greatly held back its use, is its tendency to produce increased sensitization of the heart with fatal

<sup>1</sup> Internat. Abstr. Surg., August, 1955; published in Surg., Gynec. & Obst., August, 1955.

ventricular fibrillation. A fairly long discussion on "intravenous anaesthesia" is included. Here we read of the great success of "Evipan", of the brevity of its clinical action, and of its suitability for minor operations of short duration. We also read of the introduction in 1934 by John Lundy into the Mayo Clinic of the technique of intermittent intravenous administration of thiopental sodium. This is described as having been widely used in recent years in conjunction with the muscle relaxants, "a combination which gives not only anaesthesia, but as much muscular relaxation as may be desired". Beecher and Ford point out that the search continues for other short-acting barbiturates, for these agents cannot successfully compete in controllability with the inhalation agents until intravenous agents are made which can be detoxified or broken down in the body as rapidly as the inhalation agents can be excreted from it. Though that day is probably far off, a considerable approach has been made towards it. After mention of rectal anaesthesia comes a section on local, regional block and spinal anaesthesia. With regard to the last-mentioned, some legal and economic matters are referred to. Apparently insurance companies are in some regions refusing to protect medical practitioners against claims for damage when spinal anaesthesia is employed. Beecher and Ford ask whether the techniques employed in the practice of medicine are to be determined by competent physicians themselves, or whether this is to be done by outside pressure groups. They state that if spinal anaesthesia is not useful in proportion to its dangers, it should be eliminated by those skilled in its use. With this we cannot fail to agree, though it is true that, as Beecher and Ford state, "many competent observers believe it to be valuable and important for certain purposes". With regard to muscle relaxants, it is stated as unquestionable that they represent a truly great advance in convenience for the surgeon and the anaesthetist. Whether the introduction of these agents constitutes a great advance as judged by the severe test of death-rate comparisons, awaits the compilation of an adequate series of well-controlled cases, in which these agents are used to produce muscle relaxation for prolonged periods in major surgery. It is regarded as odd that some persons, in attempting to establish these agents, have inserted a little of this or that muscle relaxant into the middle of a surgical procedure, in which the burden of anaesthesia is carried by other agents and techniques, and then have given such brief usage equal weight in cases in which the muscle relaxants have been used for long periods. Such an attitude, we are told, can add little but confusion to an already complex situation.

The sections dealing with anaesthesia for thoracic surgery and with hypothermia must be passed over. Four remaining problems are stated—the high death rate in general, the high death rate among infants and small children, cardiac arrest and ventricular fibrillation, and the need for caution. Under the last-named heading the obviously correct statement is made that there is no right to hail as progress the introduction of agents or techniques which increase the death rate from anaesthesia.

Beecher and Ford deal with liver function and anaesthesia. They point out that in their laboratory, when the type of surgery and the pre-operative state of the liver are carefully controlled, moderate to severe post-operative dis-

turbances in liver function, as judged by certain tests, have been found following anaesthesia and surgery. Evidence of liver damage has varied with the nature of the surgical procedure, and especially with the initial condition of the liver. However, no difference has been found between the effects of ether, cyclopropane, and spinal anaesthesia in patients with normal pre-operative liver function, or between those of ether, cyclopropane and hypotensive spinal anaesthesia in patients with pre-operative liver disease. It is pointed out that in a series of more than 125 patients with severe liver damage who were carefully studied, it had not been possible to demonstrate any significant difference in the clinical outcome as far as the liver was concerned, following ether as opposed to cyclopropane or hypotensive spinal anaesthesia. This statement follows one on the observation of metabolic acidosis in cirrhotic patients who had been subjected to ether anaesthesia.

An important section deals with the mechanism of action of anaesthetic agents. Beecher and Ford here quote Lillie, who pointed out that the problem of the general nature of anaesthesia was in fact inseparable from the wider problem of the nature and conditions of irritability in general. The authors point out that the irritability of the cell, why it exists and what factors influence it, constitute the central problem of all biology, of life itself. This is an adequate reason for the study of the action of anaesthetic agents. The several theories which have been advanced in the past in this connexion are set out, and finally we read that the burden of evidence indicates that the adsorption of the anaesthetic agent at the cell surface or at intercellular structures influences the metabolic activities of the cell. The nature of the lipoids present controls to a considerable extent the quality, amount and site of adsorption. At the same time, experiment has not yet shown what specific metabolic activities are affected, or what connexion exists between metabolic disturbances and changes in the functional activity. The authors admit the truth of Cushny's views, that after anaesthetic agents have penetrated into the brain cells or perhaps the environment of brain cells, the anaesthetic effects depend on some other quality which is still unknown. They conclude with the somewhat devastating remark that at the end of more than 100 years of active experience with the anaesthetic process, it remains only a little less of a mystery than it was at the beginning.

This leads to the final consideration. Study in the advances made in anaesthetics fully justifies the view that an anaesthetist is a specialist. It has sometimes been suggested by the friends of anaesthetists, not by their enemies, that they tend to take themselves too seriously. If they give this impression, it is probably the result of the struggle which they have had for recognition as specialists. While medical practitioners in general will accord the title of specialist anaesthetist to those who devote their whole attention to the subject and are skilled in it, they will expect anaesthetists to deal with the whole subject of anaesthetics—that is to say, they must not be content to practise their art for the benefit of patients and surgeons; they must also do what they can to further research in the subject. If they regard themselves as practising pharmacologists, they will the more readily do this, either in their corporate body or as individuals.



## Current Comment.

### CARDIAC REPAIR UNDER CROSS CIRCULATION.

FROM the early days of medical experimentation man has attempted to transfer the vigor of health from the young and quick to the aged and to the dying sick. Cross transfusions from animal to man were distinguished neither by success, nor by obvious disaster, probably because they were ineffective. However, these bizarre demonstrations did pave the way for the storage of blood from human donors and the modern controlled transfusion of this liquid life into the veins of the needy. Corneal grafting, skin grafting and the transfer of other active tissues are now regularly performed. Not until very recently has the active vitality of the living healthy heart and lungs been utilized to keep alive a patient whose heart has for a time been isolated from the vascular circulation. C. W. Lillehei, M. Cohen, H. E. Warden, N. R. Ziegler and R. L. Varco<sup>1</sup> have described the methods and results of the direct vision closure of ventricular septal defect in eight patients by means of cross circulation of blood from a donor. The cross circulation involved the simultaneous exchange of arterial and venous blood between patient and donor. A pump was used in the system, so that the amount of blood reaching the patient could be carefully controlled. This method of providing a continuous cardiac by-pass during operation means that open cardiomy is possible, and that the time available is adequate for proper repair once the interior of the heart has been exposed. The authors suggest that the same technique may well be applied to the repair of other intracardiac defects, such as the tetralogy of Fallot and *atrio-ventricularis communis*. All patients in this series in whom repair was undertaken were five years old or younger.

Anæsthesia was used for both patient and donor. In the former, intravenously administered "Pentothal" and curare were used once unconsciousness had supervened. After exposure of the patient's heart, cannulation was effected through a jugular vein into the inferior *vena cava*, thus providing an efficient drain of all venous blood. Arterial blood reached the patient through a cannula inserted from the right subclavian artery into the ascending aorta. Venous blood reached the donor through a catheter via a great saphenous vein, into an iliac vein, or into the inferior *vena cava*. Arterial blood was removed from the donor by a catheter through a superficial femoral artery into the abdominal aorta. Heparin was administered to both patient and donor prior to operation. The pumping mechanism was exerted indirectly onto the plastic conducting tubes, so that the extracorporeal circuit was reduced to a minimum. Body temperature was maintained at the normal level during operation, to avoid damage to the cardiac conducting mechanism. Blood flow was at 30% of the normal cardiac output for that patient. Blood loss of the patient was made up by subsequent saphenous transfusion, and there was no effective loss in the donor.

All patients selected for this method of treatment had severe signs of congenital cardiac abnormality. There were growth failure, dyspnoea, reduced exercise tolerance, cardiac decompensation and repeated episodes of pneumonia. All had cardiomegaly. Seven of the patients had severe pulmonary hypertension, as found by cardiac catheterization. Donors were selected very carefully, and in all but one case were parents of the particular children. They were rigorously examined for general disease, and it was preferred that there should be no previous history of blood transfusion. All had to be ABO compatible and of similar blood cell types, and the possibility of immunization was carefully considered in each case. Saline, centrifugal tube cross-matching was performed, as well as an indirect Coombs test.

During the operation the donor was hyperventilated with 100% oxygen. The patient's lungs were not used, but were allowed to collapse away from the operation field. The extracorporeal circulation was first charged with isotonic

dextrose. The authors describe the surgical technique of the suture and closure of the septal defect under direct vision. The right ventricular cardiomy incision was repaired, and the patient's lungs were re-inflated. Dextrose solution (5%) was used to fill the cardiac chambers and thus avoid air embolism.

In this series of cases the circulating by-pass was in operation for from five to twenty-six and a half minutes. Two of the patients subsequently died from pulmonary complications. Of four patients subjected to cardiac recatheterization some months after operation, all were found to be maintaining closure of the shunt, and in two of them pulmonary pressure had already returned to normal levels.

### SALICYLATE POISONING IN CHILDREN.

THE small child reacts to finding a strange object, especially if it resembles a sweet, by placing the object in his mouth. Hard metals and stones are usually rejected, but the infant sometimes has a surprising tolerance to both tablets and liquids which are obnoxious to the adult palate. Young children are notoriously capable of procuring boxes and bottles from seemingly inaccessible retreats, and some parents are surprisingly careless in the storage of poisonous materials, especially those prescribed for medical use. Children are found with their mouths full of half-chewed barbiturates, sulphonamides, antihistamines and salicylates, or reeking with alcohol or other toxic hydrocarbons. Some are discovered only when severe corrosion due to liniments or household fluids has already taken place. F. W. Clements<sup>1</sup> has recently written on the subject in this journal.

Probably the most common agents of accidental poisoning in childhood are methyl salicylate (oil of wintergreen) and aspirin. During a period of six months in 1952 there were 137 children under the age of five years treated in New York municipal hospitals<sup>2</sup> for poisoning by a variety of agents. Of these children, 11 were suffering from the effects of poisoning by salicylates. In the United States of America during the year 1951, out of 89 deaths caused by salicylates, 72 were in children under the age of five years. The problem of salicylate poisoning in childhood is described by A. C. Erlanson and E. C. Burke, who discuss the incidence of such tragedies and the treatment of the poisoned child.<sup>3</sup> Salicylates are rapidly absorbed into the body, being detectable in the urine within fifteen minutes of ingestion. In the body tissue 20% of the salicylate is oxidized, the remainder is excreted in the urine. The peak of blood concentration is reached within two hours, and normally 70% is excreted within the first twenty-four hours. The rate is diminished in the presence of dehydration or acidity of urine. One teaspoonful of methyl salicylate is roughly equivalent to 12 aspirin tablets.

Signs of salicylate poisoning are nausea, vomiting, hyperpnoea (mostly an increase in respiratory volume), sweating, delirium and coma. The initial respiratory stimulation leads to a decrease in carbon dioxide concentration and an increase in the serum pH value. In the case of infants and young children, initial respiratory alkalosis is followed by metabolic acidosis, and this in turn causes further hyperpnoea. Thus incorrect therapy at the particular stage of tissue metabolism is likely to lead to a fatal conclusion. Erlanson and Burke suggest that treatment of the poisoned child, if commenced within the first few hours, should commence with thorough gastric lavage. Oxygen is administered, in severe salicylism, to combat tissue anoxia as well as the hyperpyrexia which may follow salicylate intoxication. To prevent possible hemorrhage due to hypoprothrombinæmia the authors suggest that vitamin K should be given to the child in a dose of about one milligramme of vitamin K per gramme

<sup>1</sup> M. J. AUSTRALIA, March 5, 1955.

<sup>2</sup> M. Fraenkel and C. Erhardt (1955), "Morbidity in the Municipal Hospitals of the City of New York", Russel Sage Foundation, New York.

<sup>3</sup> Proc. Staff Meet. Mayo Clin., September 21, 1955.

<sup>1</sup> Surg., Gynec. & Obst., October, 1955.

of salicylate. Barbiturates, paraldehyde and morphine should not be administered, as they potentiate the toxic effects of salicylates. It is also suggested that blood should be taken from the patient and examination made for total carbon dioxide content, hematocrit reading and serum pH level. A solution of glucose should be given by the intravenous route to counteract ketosis; at the same time dehydration is corrected. The pH of the serum should be used as a guide for alkali therapy. Respiratory alkalosis gives way to metabolic acidosis within one to six hours, and the time at which the change takes place is in inverse proportion to the amount of salicylate ingested. During the period of respiratory alkalosis, isotonic saline in 5% glucose solution is given by the intravenous route, and in the stage of metabolic acidosis, sodium lactate or sodium bicarbonate is added to the infused mixture. Death when it occurs is due to circulatory and respiratory failure.

Prevention remains, of course, the most important part of the answer to the problem of poisoning by salicylates or any other agent—and it is a very real problem. The pitiful combination of the agonized parent and the poisoned child is a familiar paediatric emergency. There is no easy way to prevent accidental poisoning in young children. Parents should be warned not to store medicines unnecessarily, but the only real answer to such problems is the exercise of constant and unmitigated vigilance.

#### DOWNWARDS ESCAPE FROM JET AIRCRAFT.

The jet-propelled aircraft has already written history across the sky. The use of large passenger planes flying swiftly and smoothly at high altitudes is already in the near future. The first jet engine, as originally conceived and designed by Sir Frank Whittle, was applied to fighter aircraft and came into valuable service at the end of the second World War, and was eventually adapted for the use of the multi-engined bomber. Now all modern aircraft, designed for offensive or defensive combat, are propelled in this way. The development of these machines has raised a number of problems in human physiology and in human behaviour under the stress of high speed and high noise level, of tremendous acceleration and deceleration and of profound rapid changes in atmospheric conditions. The modern aircraft is very costly and highly complex. It is designed to function without failure, but its weakness lies in its complexity; so that under the particular conditions of high-speed, high-altitude flying, even a short-lived mechanical fault is often beyond correction and the aircraft plunges helplessly to its destruction.

Modern pilots are also highly complex repositories of expensive training and experience, and of all military personnel, without exception, are the least expendable. The ability of man as a machine is limited by his own inflexible reaction time, and slight errors are rewarded by disaster. When the crash is unavoidable, then the salvageable centre is the man and all efforts are made to ensure that he survives. However, the high aircraft speed and the shortage of time available for escape render the old methods of jumping to safety quite impracticable. The Martin Baker ejection seat was first evolved to hurl out the pilot from his crashing aircraft, to blow him, by explosives under the seat, well clear of the plane, and then automatically to ensure a safe parachute descent. This method of escape has been adapted to most military jet aircraft. Many lives have been saved, and recently the ejection seat has been adapted to ensure survival should aircraft failure occur even at ground level.

The downward ejection method of escape was more recently developed<sup>1</sup> to provide aircraft designers with an alternative method of arranging vertical emergency escape apparatus when upward ejection is impracticable. Conventional upward ejection may be imperilled by particular features of an aircraft, such as an unusually large vertical stabilizer, which would prevent safe clearance of the seat and pilot at some ranges of the aircraft. Also in bomber

aircraft, where part of the crew is stationed at the lower level, the only method of escape by mechanical vertical ejection is downwards, while the upper deck crew escape vertically upwards. One disadvantage is the inadaptability of the downward method for escape at very low altitudes.

A straight duration curve for human tolerance to negative acceleration was first constructed by Shaw in 1947. The results of these studies stressed the desirability of transmitting a large portion of the ejection load to the pelvis by means of a lap belt, as the compression load of the thoracic vertebrae is limited to that under a force of 13 G. The lap belt was designed to counteract downward acceleration and forward thrust, though there is still some uncontrollable upward thrust of the viscera against the diaphragm. As a result of the theoretical findings, a practical study was made by the use of a specially designed test tower, and further stabilizing stages were added. In addition to exerting uniform stability through the thighs and shoulders, the feet were held above the ankles and thus windblast was unable to hyperabduct the thighs with resultant dislocation of the hips. The arms were held by voluntary hand grip at calf level in the firing position, and it was appreciated that if the grip was released prematurely windblast might be dangerous. No important physiological changes were noted during the test tower experiments.

More recently actual downward ejection escape experiments have been made from aircraft flying both at high speeds and at high altitudes, human volunteers being used at the Elgin, Florida, base of the United States Air Force.<sup>1</sup> The ejection seat was installed in the bombardier-navigator position in the nose of a B-47 aircraft. The subjects were connected to the aircraft's communication system by the usual leads, but not to the oxygen system. The descents were made into the sea and elaborate precautions were taken for detailed recording of the events and subsequent rescue of the subjects. The parachutes were of standard automatic opening back style connected to the automatic opening lap belt. The parachute was opened approximately six seconds after ejection, opening being initiated by the separation of seat and subject. Delay in opening could be arranged if escape occurred at extreme altitudes. For all but the slowest aircraft ejections a partial pressure altitude suit with pressure helmet was used. Subsequent experience taught that the semi-rigid leather helmet was usually blown off at high speeds. Each of the preliminary tests was conducted from an altitude of 10,000 feet. Seven ejections were made during October, 1953. Some delay in separation of subject and seat caused delay in the opening of parachutes. Two subjects, who were ejected when the aircraft speed was 389 knots, both sustained serious arm and shoulder injuries due to loss of hand grip, with resultant windblast of the falling arms. There was also some failure of the leg guards. It was discovered that at high speeds loads upon the wrist of about 400 pounds were being exerted during the short time when the subject was falling through the slipstream of the aircraft. An elastic shock-absorbing device was constructed so that there was spreading of the wrist load, and at the same time the gripping apparatus was made more easy to hold. By these means the load was reduced below 300 pounds at aircraft speeds of 425 knots, and outward extension of the elbows was avoided.

Human volunteers were ejected during July, 1954, and this time the wrists were loosely stabilized with a nylon band, and the knees were similarly held near together to prevent excessive spreading of the legs. A modified oxygen mask and helmet were worn. Eight subjects were ejected at a height of 10,000 feet from an aircraft moving at speeds of from 303 to 423 knots. There were no major mishaps, though there were still delays in separation of the ejector seat and several subjects lost helmet and mask. The only injuries were a cut forehead and several stiff necks.

Two final ejections of human volunteers were made at an altitude of 45,000 feet. Automatic parachute opening was arranged for 11,000 feet; in addition the subject was

<sup>1</sup> W. S. Rothwell and E. G. Sperry, "Escape from Aircraft by Downward Ejection", *J. Aviation Med.*, August, 1953.

<sup>1</sup> E. G. Sperry, H. P. Nielsen and I. M. Barash, "Downward Ejection at High Speeds and High Altitudes", *J. Aviation Med.*, October, 1955.



instructed to operate manually the parachute release mechanism after falling for one hundred and fifty seconds. Subjects were connected in the standard way to the aircraft oxygen system and were equipped with a standard emergency supply for the fall. Once the parachute was open, the subject was to fire red smoke grenades to aid in tracking and to demonstrate his consciousness. In both cases the descent was successful. One subject had conjunctival and left tympanic petechiae with transient slight intellectual slowness and some residual temporary vertigo. The other volunteer had no clinical abnormality. The physiological damage in the first man was due to excessive free spinning prior to opening of the parachute, and this, combined with unpredictable separation from the ejection seat, provide the two main hazards of downwards ejection still to be overcome.

While men continue to seek for further victories over the forces binding them to the earth, other men must reach out and move ahead in their gigantic game with death. These brave people tumble through the sky and are lost in the oblivion of statistics. They will not be forgotten by those who fly after them in the reassurance of safety.

#### NUCLEAR EXPLOSIONS AND FALL-OUT RADIATION.

MANY words have been spilled onto paper in feverish attempts either to justify or to bring about the elimination of nuclear weapons. In the meantime the shifting moodiness of nations and the suspicions of power politics appear to necessitate continued research into, and publicized demonstrations of, more powerful weapons of mass destruction. Reassurances have always been given that there was no risk of appreciable exposure to radioactivity in the innocent who inhabit the remote regions chosen for the experiments. The severe contamination of Japanese fishermen after one such atomic explosion first created some uneasiness in the minds of some who wondered whether the experiments were so accurately controlled after all. Above the farthest cities radioactivity increases detectably as each great power throws its latest bomb. The effects upon gene patterns are well known, and the relative indestructibility of much radioactive contamination is notorious.

E. P. Cronkite, V. P. Bond, R. A. Conard, N. R. Shulman, R. S. Farr, S. H. Cohn, C. L. Dunham and L. E. Browning write of the finding of changes in human beings accidentally exposed to atomic radiation. In the spring of 1954 a nuclear device was exploded in the Marshall Islands. A few hours after the explosion a white snow-like material settled from out of the sky, clinging to hair and skin of the native inhabitants and infiltrating into their dwellings. This material consisted of pulverized and incinerated coral (calcium oxide) coated with radioactive fission products which had become disturbed by winds high in the atmosphere. Living on the island most severely affected were 64 Marshallese natives, who received an average calculated whole body dose of 175r, in addition to skin contamination, inhalation and ingestion of radioactive material. Within a few hours two-thirds of the natives developed nausea, which lasted for two days, while 10% developed vomiting and diarrhoea. Itching and burning of the skin and burning and lachrymation of the eyes were soon noticed. There was no supervised decontamination until well after the initial period, and an emergency medical team arrived on the ninth day after exposure.

Serial determinations of the levels of blood cells were carried out and compared with similar groups of natives of the same ages. Results showed that fluctuations which occurred in the total leucocyte count were due largely to variations in the concentrations of neutrophilic cells. The neutrophilic cell counts fell to about 75% of control levels in the second week, fluctuated until the fifth week, and

then a second depression to about 50% occurred, followed by gradual return to 75% of normal. It was noted that the younger age groups were more severely affected. Lymphocyte cell concentration in the older age groups fell to 55% by the third day and improved very little thereafter. In the younger age groups the lymphocyte concentration fell to 25% of normal, but thereafter showed a very gradual recovery.

No serious infection occurred, despite the fact that 27 of the natives developed neutrophilic cell counts of less than 2500 per cubic millimetre, and temporarily levels of 700 to 1000 were found. Platelet counts in women at the tenth day were 60% of normal, and gradually fell to 30% at the fourth week. After this time the count gradually rose again to 70% at the seventh to eighth week, remaining at this level. It was noted that 20% of the population, at the fourth week, had less than 90,000 platelets per cubic millimetre of blood, but no tissue haemorrhage was found in any of the natives.

Follow-up studies after six months revealed little improvement in the level of blood cells; but after a year the levels of neutrophilic cells were normal. However, lymphocytes, monocytes, eosinophilic cells and platelets still remained at subnormal levels. Observations revealed little change in the concentration of circulating erythrocytes, apart from some temporary depression at about the twenty-second day after exposure.

Skin lesions occurred in 90% of the natives, and were due to  $\beta$  radiation from fall-out deposit. Changes, which included epilation, began at twelve to fourteen days after exposure and were modified or absent in those under shelter, by the wearing of clothing, and by bathing. Scalp lesions usually preceded epilation and were more pronounced in children. Normal regrowth began at nine weeks and was complete within six months.

No primary or secondary erythema was observed in skin lesions, and latent periods for the appearances of changes were irregular. The neck and scalp were the most commonly affected areas, but many lesions of the ante-cubital fossae and the feet were found. The first skin change observed was a local increase in pigmentation with macules, papules and raised plaques. After several days dry, scaly desquamation took place from the centre outwards, leaving depigmented but otherwise normal skin. Repigmentation usually took place in the succeeding few weeks. It was found that 20% of skin lesions were more severe, particularly on the neck, scalp, ears and feet. These were painful lesions with wet desquamation, with weeping and crusting, and in some instances with bullous formation. Healing, with one exception, took place within ten days. Repigmentation tended to be excessive, and the healed areas were thickened; but follow-up studies revealed that the pigmentation gradually returned to normal. The skin of severely burned areas remained unpigmented and atrophic.

Radiochemical analysis of urine samples revealed that internal radiation dosage was probably negligible. There was some general weight loss in the natives during the period of observation. Four pregnant women subsequently gave birth to normal infants.

Thus for the first time there has been the accidental exposure of a large number of innocent bystanders to radioactivity. The dose received by the Marshallese natives most affected bordered on the lower fatal limits. By good fortune there were no deaths, though several children were severely affected by the radiation. The long-term effects of the accident cannot be calculated. Any resulting gene disturbance will already be irreversible, and the unfortunate individuals, especially the scarred children, may well succumb at some future date to malignant changes. These people, and their less affected neighbours, will be carefully observed; in some horribly calculating way they may even be classed as a closed human experiment.

## Abstracts from Medical Literature.

### RADIOLOGY.

#### Rapid Examination of the Biliary Tract with "Biligradin".

N. H. ALDRIDGE (*J. Fac. Radiol.*, April, 1955) describes a method of examining patients by means of "Biligradin" which is based on the results of experimental evidence. He states that in this way, at a single attendance, a complete cholecystogram and cholangiogram may be obtained within ninety minutes in an average case. Unless the patient tends to be constipated, no special preparation is used. Where needed, one ounce of castor oil may be given at six o'clock on the preceding evening. On the morning of the examination the patient omits breakfast, taking instead half a "Felevec" fatty meal. At the examination control films are taken; then 25 to 50 milligrammes of pethidine hydrochloride are given intravenously, followed by 40 millilitres of "Biligradin". If the pethidine and "Biligradin" are mixed, a dense white insoluble precipitate forms; thus the two cannot be given in the same syringe. To avoid two separate injections, a simple method of using two syringes with one needle has been used: a small syringe is used for the pethidine, and after this has been injected slowly, the plunger is drawn back, so that venous blood enters the barrel of the syringe. This is then returned to the vein, and the procedure may be repeated once or twice. The first syringe is then disconnected, the needle being left in the vein. The larger syringe containing "Biligradin" is then connected to the needle, and the second injection is given very slowly. The "flushing" of the needle before the "Biligradin" is given may also be accomplished by using saline, but the above method is less tedious. A film is exposed twenty minutes after injection of the medium to show the ducts, with the patient in the prone position and the right side raised 30° from the table. A large cone is used for this film; but when the ducts have been localized, the smallest possible cone is used. The second film is taken with the patient in the prone position at forty-five minutes to show the gall-bladder, and an erect film is also taken at this stage. If the gall-bladder is adequately filled, the second fatty meal is given and a final film taken twenty minutes later to show contraction of the gall-bladder. Intermediate or later films may be taken as required in particular cases. Of a series of 100 unselected patients examined by this method, gall-bladder visualization was obtained in 97% and common bile duct visualization in 100%.

#### Osteopetrosis in Adults.

C. L. HINKEL AND D. D. BEILER (*Am. J. Roentgenol.*, July, 1955) review the clinical and radiological findings of osteopetrosis in adults. They state that in adults the manifestations are different from those of the better known infantile form. The course is clinically benign,

and hematological and neurological findings are minimal or absent. Long-bone clubbing, medullary narrowing and foramen encroachment are not prominent. The disease may be asymptomatic. The disease probably develops as the result of an hereditary trait which is governed by one or more genes of intermediate dominance. The essential defect appears to be inherent in the mesenchyme, which is the common ancestor of hematogenic and osteogenic cells. In siblings the type and distribution of the bone changes are highly characteristic of the family. However, there may be considerable variation in the time of onset and in the severity of the disease. The increase in density results from faulty resorption of endosteal and periosteal bone and matrix. Cartilage cells and unresorbed matrix are found in the bones of adults as late as the seventh decade. Osteopetrotic bones are increased in weight, fragility and mineral ash content. In patients of this age the dense inclusions are probably not viable. It is the authors' impression that although exacerbations and remissions may occur, when the osteopetrotic inclusions have been enveloped by normal bone the disease is no longer active.

#### Congenital Abnormalities of the Feet.

L. A. DAVIS AND W. S. HATT (*Radiology*, June, 1955) discuss congenital abnormalities of the feet. They state that since at birth the talus and calcaneus are the only tarsal bones which are ossified, the anatomical analysis consists essentially in a description of the relationship of these two bones to each other and to the metatarsals. The technique for obtaining antero-posterior and lateral skiagrams must be carefully standardized and followed. Slight variations in rotation in either projection can greatly alter the relationship of the bones as shown on the film. For the antero-posterior view the knees must be held together and fall in a plane which is perpendicular to the film. The tendency of the technician to "correct" the abnormality by placing the foot "normally" on the cassette must be discouraged. For the lateral projection the technique for a lateral ankle view is the correct one. In this position, rotation of a small degree will result in a change in the apparent relationship of the talus and calcaneus.

#### Pulmonary Ossification in Patients with Mitral Stenosis.

W. WHITAKER, A. BLACK AND A. J. N. WARRACK (*J. Fac. Radiologists*, July, 1955) describe seven patients with mitral stenosis complicated by pulmonary ossification and discuss, in the light of recent observations, the evidence relating pulmonary ossification to abnormal pulmonary blood pressure in patients with mitral stenosis. They state that the present series confirms previous observations that small intraalveolar nodules of bone occur in the lungs of patients suffering from mitral stenosis, and that these nodules are found predominantly in the lower lobes of the lungs and do not involve the apices. The opacities which these nodules of bone produce on radiological examination

must be differentiated from those which occur in patients with pneumonokoniosis, miliary tuberculosis, sarcoidosis, leukaemia, Hodgkin's disease, lymphangitis carcinomatosa and infection with *Histoplasma capsulatum*. Such a differentiation presents little other than a theoretical problem once the presence of mitral stenosis is recognized, since the association of these other conditions with mitral stenosis must be extremely rare. Any patient with mitral stenosis and pulmonary ossification will also have at least histological evidence of hemosiderosis, but ossification is not a sequel of pulmonary hemosiderosis. It is believed that both complications are the direct result of abnormal pulmonary vascular blood pressures, and that hemosiderosis is produced by small recurrent broncho-pulmonary vascular hemorrhages while ossification occurs in the fibrinous exudate of pulmonary oedema. The concept of a fibrinous alveolar exudate as the site of origin of ossification is in keeping with the histological appearances, but there is still uncertainty about the factors responsible for the production of this exudate and its subsequent ossification. Although it is apparent that pulmonary ossification occurs in patients with mitral stenosis who have abnormally high pulmonary blood pressures, which predispose them to attacks of pulmonary oedema, there are still reasons why it is difficult to accept these complications as the primary cause of pulmonary ossification. Only two patients in the present series had symptoms of "left ventricular failure", which indicates that if pulmonary oedema is a precursor of pulmonary ossification, it may not necessarily be acute generalized pulmonary oedema. However, just as hemosiderosis may develop owing to bleeding from broncho-pulmonary vascular hemorrhages in patients having no history of gross haemoptysis, so it seems possible that localized pulmonary oedema may occur in patients who never have symptoms from this complication. Although such localized zones of pulmonary oedema may be sites of subsequent pulmonary ossification, factors other than abnormal pulmonary blood pressures must be involved in the production of the osseous nodule, since such pressures are common in patients with mitral stenosis while pulmonary ossification is not.

#### The X-Ray Signs of Carcinoma of the Lung.

L. G. RIGLER (*Am. J. Roentgenol.*, September, 1955) discusses the differential diagnosis of pulmonary carcinoma. Great importance is attached to the demonstration of the presence or absence of calcium in the mass. It is stated that such a determination is best made by laminagraphy; the procedure is highly effective in demonstrating calcium. While it is true that the absence of calcium is of little significance, since many granulomata and benign lesions do not show calcium, the presence of calcium is of great significance. A few cases, both of bronchial adenoma and of carcinoma, have been reported in which calcium was demonstrated in the lesion. In some of these the calcium may well have been present prior to the



development of the tumour, which happened to occur in the same area. In others necrosis of the tumour resulted in calcification. Nevertheless, these are startling and extraordinary exceptions to the general rule. For practical purposes, therefore, the presence of calcification, barring compelling evidence to the contrary, should indicate that the lesion is not a malignant tumour. The author also describes a sign which he regards as characteristic of malignant change. This consists of a notching or umbilication in the margin of the shadow. The deformity is not well seen in the ordinary skiagrams, but it is quite clearly delineated in body section skiagrams when the film is made at the proper level. The cause of this notching is not entirely clear, although in several sections it appears to resemble a hilus in the mass containing blood vessels and normal lung structures extending into the body of the mass itself, thus producing the irregularity of the contour. The notching is usually single, although in some cases it has been multiple. The importance of this notch is emphasized in determining that the patient has an actual malignant condition. As yet it has not been seen in any benign condition.

#### PHYSICAL THERAPY.

##### Carcinoma of the Prostate Treated with Radioactive Gold.

H. D. KERR, R. H. FLOCKS, H. B. ELKINS, D. CLUP AND T. C. EVANS (*Radiology*, May, 1955) report a series of 100 cases of carcinoma of the prostate treated by combined interstitial injection of radioactive gold and surgery. They state that the cases treated in this manner are those in which local extension has made radical surgery impossible, but in which distant metastases could not be demonstrated. Exploration is carried out in all cases by a retropubic approach, the size of the gland is estimated, and a search is made for metastatic nodes. Any grossly involved lymph glands are removed, and in some cases a large proportion of the primary tumour is removed as well. The radioactive gold is then injected into the tumour. "Urokon" (70% solution) is added to the injected material, so that subsequent radiographic examination will give details of the distribution of the radioactive gold. Follow-up examinations are made at frequent intervals, and if suspicious nodules are felt *per rectum*, these are injected perineally with more radioactive gold. In some cases, four or five injections have been carried out, and all signs of persistent neoplasm may disappear. Among the first 50 patients, seven developed rectal ulceration, and four of these required colostomy. In the next 50 cases there have been no rectal complications. This complication has been avoided by a refinement of technique. Of the 100 patients observed for from twenty-four to thirty-two months, 46 were still alive, 18 with obvious carcinoma and 28 apparently normal, while 54 were dead. Experience has shown that it is impossible to sterilize any except the smallest of neoplasms

with radioactive gold. The authors have therefore resorted to surgical removal of the tumour before or after injection of Au<sup>198</sup>. However, 28% of patients treated by this method had no clinical evidence of neoplasm over a period of observation of at least two years.

##### Combined Parenteral "Aureomycin" Therapy and Irradiation for Far Advanced Cancer.

J. C. BATEMAN, C. P. DONLAN, C. T. KLOPP AND J. K. CROMER (*Am. J. Roentgenol.*, July, 1955) state that intra-arterial administration of "Aureomycin" in large doses combined with nitrogen mustard had been found to improve the operability rate in cases of advanced carcinoma. Because of this fact, it was decided to combine antibiotic therapy with irradiation, and follow with surgery if possible, in selected cases of advanced malignant disease. "Aureomycin" hydrochloride buffered with sodium glycinate was given by a regional intraarterial route wherever possible. In cases of abdominal or pelvic carcinoma, the abdominal aorta was cannulized and the drug administered twice daily to in-patients and once daily to out-patients. Irradiation was given in most cases as external X-ray irradiation, but intracavitary radium therapy was employed for four of the cases of carcinoma of the uterus. The tumour doses delivered in most cases were less than the amount generally considered necessary to produce a favourable clinical response; yet in three cases no residual cancer was found at subsequent operation. This observation agreed with the clinical impression that the sensitivity of the tumour had been increased. It is emphasized that the majority of these cases were such that under ordinary circumstances irradiation would not have been considered even as a palliative measure. Clinical improvement was noted in 32 patients of 39 treated in this manner. Of these, nine showed no evidence of disease four to nineteen months after treatment was started. The authors feel that the results, although not impressive, are encouraging enough to warrant further trial of this method of combined therapy.

##### Two-Million Volt X-Ray Therapy.

R. J. GUTTMANN (*Am. J. Roentgenol.*, August, 1955) gives a brief report on 40 patients who were treated with two-million volt X-ray therapy for various malignant lesions of the upper part of the abdomen at the Francis Delafield Hospital, New York, between August, 1951, and October, 1953. There were 19 cases of carcinoma of the liver, two of hepatoma, six of carcinoma of the stomach, two of carcinoma of the common bile duct and a number of other types. Of the 21 patients with malignant disease of the liver, six were in the terminal stages of the disease and died during treatment, eleven showed improvement in relation to pain and pressure symptoms, and six showed shrinkage of the mass. The improvement lasted for from two months to one year. Of six patients with carcinoma of the stomach, two showed gratifying results, and are living and well, doing normal work nine months and eleven months respectively after

treatment. One patient with recurrent carcinoma of the caecum and metastatic glands is alive and well with no evidence of disease two and one-half years after treatment. Another who was referred with a large inoperable liposarcoma, which measured 18 by 14 by 8 centimetres and had invaded the stomach and pancreas, had no symptoms and no evidence of active disease nine months after treatment. The results are regarded as encouraging and gratifying, in what is ordinarily an unrewarding group of cases.

##### The Hazards of Not Using Radiotherapy.

AN EDITORIAL (*Radiology*, May, 1955) states that many physicians are aware of the fact that there is a small group of benign lesions in which irradiation is the therapeutic method of choice. These will respond to doses that will leave no harmful late effects. The most important of these are as follows: naso-pharyngeal lymphoid hyperplasia which is producing symptoms not controllable by simpler means; acute post-operative parotitis in patients resistant or allergic to antibiotics; *fibromyoma uteri* in females over the age of forty years, with symptoms therefrom; pituitary disorders with related endocrine dysfunction; ankylosing spondylitis with symptoms; axillary furunculosis and certain other infections or inflammatory disorders of the skin and its appendages, notably *acne vulgaris*; *tinea capitis* due to *Microsporon audouinii* infection; tuberculous cervical adenitis; hyperthyroidism; subacute thyroiditis; selected ophthalmological disorders, notably pterygium, vascular scars and dendritic keratitis. These widely diverse conditions are sometimes a source of serious disability, and in suitably selected cases can be relieved or permanently cured by irradiation. It is important that the efficacy of irradiation in these conditions should be recognized; the generalization that radiotherapy should be reserved for malignant conditions, which is sometimes expressed, means that many patients would lose their best opportunity of relief.

#### SURGERY.

##### The Treatment of Duodenal Ulcer by Vagotomy and Gastro-Jejunostomy.

R. L. HOLT AND R. F. ROBINSON (*Brit. J. Surg.*, March, 1955) consider that subtotal gastrectomy can be criticized as being too drastic a method of curing duodenal ulcer, particularly in young patients. They report the results that they have obtained from a series of 243 patients operated on between 1947 and 1953. Subdiaphragmatic vagotomy was carried out in a manner similar to that described by Dragstedt in 1945, and posterior, horizontal isoperistaltic gastro-jejunosomy was performed. The mortality was low (one death in 243 cases), and the results were such that the authors consider that this method of treatment is worthy of serious consideration. It has a low mortality, an absence of serious post-operative complications, and a high proportion of satisfactory results.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at Bendigo on Saturday, July 16, 1955.

#### Clinical Meeting.

At the afternoon session at the Bendigo Base Hospital a series of patients was presented and discussed.

#### Cyst of Gastro-Colic Omentum.

The first of five patients presented by Dr. A. L. NEWSON was a married woman, aged fifty-nine years, who had been first examined on May 6, 1955, when she was complaining of a burning feeling in the epigastrium which was relieved by antacids. A large cystic mass was detected in the epigastrium. At laparotomy on May 20 a simple cyst the size of a large grapefruit was removed from the gastro-colic omentum, and the report on a section of the cyst wall was "simple serous cyst". Dr. Newson said that omental cysts were rare and were presumably caused by obstruction of lymphatic channels. They were best treated by enucleation rather than by excision of surrounding omental fat.

#### Ulcerative Colitis with Ileostomy and Partial Colectomy.

A man, aged forty-one years, had been first examined in April, 1954, when he was complaining of bloody diarrhoea and cramp-like abdominal pains present for eight months. On examination he was found to have an ulcerated mouth and pustular rash on the body. Proctoscopic examination revealed red oedematous rectal mucosa, with blood and pus in the lumen; but no ulcers were seen. The results of three barium enema X-ray examinations were negative. Subsequently he had exacerbations of the disease, and his general condition deteriorated. In June, 1954, an ileostomy of the Cattell type was performed, and in subsequent weeks the case was complicated by the formation of a huge anterior perianal abscess requiring incision and two subsequent operations to open fistulous tracks, several severe colonic hemorrhages requiring transfusions, joint involvement of a rheumatoid nature, and hepatitis with deep jaundice. In December, 1954, a partial colectomy was performed; the transverse colon and descending colon as far as the left colic artery were excised, and the cut end of the remainder of the descending colon was implanted at the left end of the incision in the transverse colon. The whole of the removed colon was grossly affected, with thickened and inflamed wall, the mucosal surface showing multiple polyp. Examination of a section of the colon wall showed "inflammatory infiltration with small abscesses beneath the mucosa". Subsequent to operation there was gradual improvement in the patient's general condition, with disappearance of the joint swelling and a gain in weight, but unfortunately there had been some sloughing of the ileostomy with digestion of wound margin followed by prolapse. The patient was now fit for completion of the colectomy and found that the use of a Davol bag was satisfactory.

In discussing the case Dr. Newson said that in the opinion of Colcock and Lahey 25% to 40% of patients with ulcerative colitis required surgical treatment, and the indications for surgery were an acute exacerbation, hemorrhage, infectious arthritis, perianal suppuration and fistula formation. It was pointed out that in the case under discussion four serious complications (perianal suppuration, hemorrhages, joint involvement and hepatitis), which occurred after ileostomy, stressed the need for early colectomy in severe cases.

Dr. G. NEWMAN MORRIS said that at a recent demonstration at the Repatriation General Hospital, Heidelberg, a plastic ileostomy bag with disposable envelopes had been demonstrated; it added greatly to the comfort of patients.

#### Gastric Carcinoma.

A married woman, aged seventy-two years, had been first examined in October, 1950, when she had a history of epigastric pain for some weeks with loose motions; her weight was then eleven stone three pounds. X-ray examination after a barium meal showed a filling defect just below the middle of the greater curvature, with an irregular margin. In December, 1950, a subtotal gastrectomy (Pólya type) was performed. Macroscopically there was a polypoid carcinoma of the greater curvature with three malignant glands in the base of the great omentum opposite the

primary growth. Microscopically the diagnosis was "carcinoma".

Subsequently the patient developed a macrocytic anemia, but was now in good health, weighing nine stone eight pounds. It was considered that the favourable result was due to the fact that the growth was apparently of an unusual slow-growing type.

Dr. L. H. BALL said that most surgeons had had one patient with carcinoma of the stomach who had survived for four years; he had one still living after seven years. He was of the opinion that gastrectomy was palliative rather than curative, and that there was not much difference in the end results following complete or partial gastrectomy; but after complete gastrectomy there were marked nutritional changes.

#### Carcinoid Tumour of the Ileum.

A man, aged fifty-three years, had been first examined in July, 1951, when he was complaining of periodic "indigestion" for four years, with epigastric pain one hour after food. His weight was eleven stone ten pounds, and there had been some slight loss of weight; but barium meal X-ray examination revealed nothing abnormal, although an elusive mass could be felt in the right iliac fossa. The patient was not examined again until September, 1951, when he had signs and symptoms of subacute intestinal obstruction, and his weight had fallen rapidly to nine stone seven pounds. After preliminary gastric suction and intravenous therapy a laparotomy was performed, and a nodular tumour of the ileum one inch proximal to the ileo-caecal valve was discovered, causing a stricture with gross dilatation of the proximal part of the ileum. Enlarged glands were present in the adjacent mesenteric angle, and there was free fluid in the peritoneal cavity. One foot of ileum, the whole of the caecum and the ascending colon were resected, and the small intestine was anastomosed "end to end" with the colon, a procedure made possible by the gross dilatation of the small intestine.

PROFESSOR E. S. J. KING gave the following report on the histological appearances of the operation specimen:

The tumour tissue is composed of masses of cells which vary considerably in size. Some of these masses are large and rounded and others are relatively small and narrow. Near the lumen some of the cell masses are in continuity with the crypts of Lieberkuhn, and in the muscle layer they lie between groups of muscle fibres. They are particularly numerous between the muscle coats and on either side of them.

Most of the cells are spheroidal in form. The protoplasm is somewhat granular and contains tiny vacuoles; the nuclei are central in position and are rounded or oval in form. The chromatin is finely reticulated and numerous small chromatin granules are scattered through it. In the larger masses there are small rounded cavities which are lined by columnar cells. These resemble the spheroidal cells in the structure of the protoplasm and the nuclei are oval. The margin of the cell masses is composed of columnar cells which are a little more elongated than those described above. Amongst these an occasional larger cell with a bulbous extremity towards the centre of the cell mass may be found (the racquet shaped cell).

The application of silver impregnation methods (Masson) shows that the cells contain numerous brown or black granules, that is, they are typical argentaffin cells. The tumour is a typical carcinoid or argentaffin tumour of the ileum. The lymph nodes showed hyperplasia of the reticulo-endothelial cells of the sinusoids, but there was no suggestion of any metastasis from the primary tumour.

Dr. Newson said that the patient had remained well since operation.

Dr. L. H. BALL said that he had recently met with a case of endometriosis in the lower part of the ileum. The patient was suffering from subacute obstruction thought to be due to a band after an earlier hysterectomy. At operation there were no adhesions in the abdomen, but one foot above the ileo-caecal junction there was a big mass, which after removal was diagnosed as "endometriosis". Subsequently the patient was treated with deep X-ray therapy, and the outlook appeared good.

#### Diverticulitis of the Sigmoid Causing Obstruction.

A married woman, aged fifty-eight years, had been first examined in April, 1955, when she had evidence of acute intestinal obstruction, supervening on chronic obstruction. For eight months previously she had suffered from diar-



rhoea, having eight to ten motions daily, with passage of mucus and occasional flecks of blood. Five days before her admission to hospital she had developed a complete obstruction following a dose of castor oil. At laparotomy on the day of her admission to hospital gross dilatation of the colon was revealed above the stricture of the lower part of the sigmoid. The sigmoid wall was thickened some distance above and below the stricture, and there was infiltration of the mesosigmoid with inflammatory adhesions to the left ovary and Fallopian tube. A transverse loop colostomy was performed, and subsequent investigation by sigmoidoscopic and X-ray examination showed an obstructive lesion two inches long in the distal part of the sigmoid.

#### *Bilateral Carpal Syndrome.*

The first of three patients shown by Dr. EUGENE SANDNER was a married woman, aged forty-six years, who had been first examined in June, 1954, when she was complaining of tiredness and irritability for the previous twelve months and recently of "pins and needles" in the left hand. Administration of "Butazolidin" gave some relief, but the pain recurred in May, 1955, when "Novocain" and "Hydrocortone" were injected into the left wrist with much subsequent improvement; about that time a similar condition developed on the right side, the pain being relieved by an injection of the same drugs. Radiological examination showed some decalcification of carpal bones, and Dr. Sandner sought advice regarding further treatment.

Dr. G. NEWMAN MORRIS said that female sex hormones were of benefit in some cases.

Dr. H. G. FURNELL said that section of the transverse carpal ligament gave immediate relief and at times produced a permanent cure.

#### *Congenital Arterio-Venous Fistula.*

The second patient shown by Dr. Sandner, a child, aged two years, had been normal at birth, and operation for a right inguinal hernia had been performed at seven and a half weeks of age. After the operation the mother had noticed that the right lower extremity was larger than the left, and since that time it had become progressively larger and more elongated. The patient was presented for discussion and advice.

Dr. D. SKEWES said that he had recently met with a similar case in a patient who had been referred to Dr. Kaye Scott, of Melbourne, and diagnosed as having an arterio-venous shunt. The advice given was that nothing should be done until the child had reached fifteen years of age.

Dr. K. MORRIS said that in the case under consideration the larger limb was much warmer than the smaller, and an arteriogram should be prepared. He considered that the child had an arterio-venous fistula, which might be localized and amenable to surgery, but might be diffuse.

Dr. J. R. MCCOY said that on examination of the child he thought he could feel a localized mass of vascular tissue posteriorly; if an arteriogram showed that surgery was practicable he would not wait until the child was much older.

Dr. M. CLARK agreed with the diagnosis of a vascular abnormality and was of the opinion that unless the condition was amenable to surgery, cardiac failure would supervene.

Dr. H. G. FURNELL referred to a case of arterio-venous shunt in which operation had been performed twenty years previously by Sir Albert Coates—the first he had performed after an arteriogram; although the patient now had many varices, she was pleased with the result.

#### *Shotgun Wound of the Arm.*

Lastly, Dr. Sandner showed a man who, in November, 1953, had been injured by the discharge of a shotgun at short range, the upper end of the left humerus being shattered. At operation on the same day fragments of bone, cardboard wads, shot and pieces of clothing were removed from the wound, which was packed with "Chloromycetin" powder. That was followed two days later by débridement and closure of the wound. Subsequently there was suppuration, and on December 1 the wound was reopened and further fragments of cardboard and loose bone were removed. On December 15, 1953, a large sequestrum was removed, and on February 15, 1954, a split bone graft (five inches of fibula) was inserted between the proximal and distal ends of the humerus (being held in place with vitallium bolts and screws), together with cancellous bone chips from the right ilium. By May, 1954, the patient had good movement at the shoulder and elbow, but early in July, after a sudden movement, the graft was fractured. On July 2 the bolts and screws were removed, the ends of the humerus drilled and cancellous chips from the left ilium inserted. By

February, 1955, union appeared firm, and the plate, which had become loose, had been removed. The patient was now back at work.

Dr. Sandner said that he had been struck by the fact that he had now met with two cases in which, despite extensive muscular and other damage, the neuro-vascular bundle had survived.

PROFESSOR RONALD HARE asked why "Chloromycetin" had been used in the wound and if the local staphylococci were resistant to penicillin.

Dr. G. NEWMAN MORRIS, commenting on Dr. Sandner's observation, referred to the case of a nursing sister who had been injured in a jeep accident during the war; the whole of the structures of the upper arm had been severed with the exception of the artery and nerve, which in some strange manner appeared to have slid out of the way.

#### *Shoulder-Hand Syndrome.*

Dr. A. J. WALTERS presented two patients. The first of these, a married woman, aged fifty-one years, was a diabetic patient with mild hypertension, who fourteen months previously had sustained a Colles fracture of the right wrist, with minimal displacement. Four and a half weeks later she complained of pain and swelling in the right thumb, spreading to the dorsum of the hand, and during the next three weeks the pain spread up the arm. X-ray examination showed decalcification at the lower end of the radius, ulna and carpus, and she was treated with calcium and vitamins and with physiotherapy. Her condition remained static for the next twelve months, with swelling and venous congestion of the dorsum of the hand and wrist, painful limitation of flexion and extension of fingers, and pain and tenderness in the forearm and arm muscles which were worse at night. Over that period treatment with calcium, vitamins, physiotherapy and "Butazolidin" had given no relief. "Ansolsen" had relieved the patient temporarily, but the best results had been obtained by the use of cortisone and hydrocortisone.

Dr. FRANK MAY suggested that contrast baths and wax baths, which the patient could carry out at home, might be of benefit.

In reply to a question by Dr. A. CLARK, Dr. Walters said that "Ansolsen" had been used as it was supposed to have a vasodilator effect at the periphery, but the benefit from it had been short-lived.

#### *Patent Ductus Arteriosus.*

The second patient shown by Dr. Walters was a married woman, aged twenty-nine years, who had presented herself eighteen months previously complaining of episodes of "palpitations" for the preceding three months and a history of shortness of breath on exertion for eighteen months. On examination she was found to be a short, obese woman. The blood pressure in the right arm was 140 millimetres of mercury, systolic, and 80 millimetres, diastolic, and in the left arm 145 millimetres of mercury, systolic, and 85 millimetres, diastolic. There was evidence of cardiac enlargement, and a systolic bruit was heard over all areas, being at its maximum in the third left intercostal space one and a half inches from the mid-sternal line with an associated diastolic "machinery murmur". Radiological examination revealed an enlarged left ventricle with hypoplastic aorta and increased hilar pulsation. Dr. Walters considered that the diagnosis was patent ductus arteriosus.

Dr. W. J. RAWLING, in discussing the question of pregnancy in patients such as the one presented, said that most cardiac patients, apparently as a compensation of Nature, had quick rapid labours; and while coarctation of the aorta might be an indication for Caesarean section, in most cases the thing to do was to treat the cardiac condition and leave the pregnancy alone.

Dr. MAURICE CLARK thought that the patient might be suffering from pulmonary hypertension in addition to a patent ductus arteriosus, as in some cases the two conditions were associated; but for some reason unknown that was not always so. Although the patient had few cardiac symptoms, he thought operation desirable and would advise it in all adult subjects because of the risk of complications as they became older.

Dr. K. MORRIS said that he agreed with the suggestion that the patient probably had pulmonary hypertension, and that the condition added to the problem of the surgeon; but blood pressure could now be controlled with hypotensive drugs given by the intravenous drip method, and the reduction of the pressure to 70 millimetres of mercury facilitated the work of the surgeon. Simple ligation of the ductus was hazardous; the duct had to be completely divided. He was

emphatic that the patient shown should be subjected to operation and was certain that after it she would be greatly improved.

#### *Beta Haemophilia.*

DR. W. ROSENTHAL presented a boy, aged nine months, of blood group A, Rh-negative, who had been diagnosed as suffering from  $\beta$  haemophilia subsequent to investigation at the Baker Medical Research Institute. He suffered from recurrent minor haematoma, usually without any apparent trauma. He had been diagnosed as haemophilic after prolonged bleeding subsequent to circumcision at the age of three weeks. At present he was undergoing a combined course of injections for immunization against tetanus, diphtheria and whooping-cough. Minor bruising had occurred after the first injection. Dr. Rosenthal pointed out that important considerations were management and advice to the parents and the desirability of sterilization of the mother. Investigation of the family history revealed that the great-grandmother and mother had had abnormal bleeding tendencies, and a second cousin had been diagnosed as haemophilic when aged nine months.

DR. R. J. SAVERS said that haemophilia was not as rare as was commonly thought. He had personally examined 76 haemophilic subjects in Victoria and the condition occurred in one in 5000 births; but of 20 severe haemophiliacs only four had a family history of the condition, the other cases having occurred by mutation. Consequently, sterilization would not do much to diminish the incidence of the condition. In one of the families he had investigated there were four male haemophiliacs. In reply to a question concerning the incidence of haemorrhage from the umbilical cord, Dr. Savers said that the condition was not common, but it did occur; if the cord was crushed with bone forceps, bleeding was not likely to occur.

#### *Obesity.*

The second patient presented by Dr. Rosenthal, a man, aged forty-eight years, a machine worker at the ordnance factory had had a severe attack of influenza at the age of twenty-eight years, prior to which he had been well and weighed nine stone. After that illness he gained weight rapidly, and by the age of thirty-two years his weight had practically doubled; it had remained at about that level. Between 1939 and 1943 he had been rejected for service in the army because of "high blood pressure", which he had been told was 190 millimetres of mercury. There was no family history of obesity. In May, 1955, he was admitted to hospital with dyspnoea associated with dizziness and swelling about the face and ankles, the attack being preceded by a "cold" with productive cough. Prior to the onset of his illness he had been regularly employed and had ridden to work each day on a bicycle without dyspnoea. His height was five feet five inches, and his weight on May 9, 1955, was twenty stone seven and three-quarter pounds. His blood pressure was 190 millimetres of mercury, systolic, and 120 millimetres, diastolic. His visual fields were normal. His urine had a specific gravity of 1030, and contained a trace of sugar but no albumin; microscopically a few pus and red blood cells were detected. Examination of blood films showed no abnormality in white or red cells; the haemoglobin value was 100%, and the red cell count 5,000,000 per cubic millimetre. The sugar tolerance curve was normal. Radiological examination showed osteoarthritic changes in thoracic vertebrae and a transverse cardiac diameter much greater than normal with congestion of both lungs. By June 5, 1955, after administration of "Neptal" and a diet of approximately 1900 Calories, his weight had fallen to sixteen stone eight pounds and had remained at that level. The patient had now returned to work and said that he felt well.

DR. HAMILTON SMITH said that the patient was a fat man with hypertension, and further investigation might not reveal anything more. It was questionable whether any treatment would help, and there were no positive clues to indicate Cushing's syndrome. The long history of hypertension was against that diagnosis, and the patient's condition did not seem to have progressed as would true Cushing's syndrome. Dr. Smith quoted three examples of the established condition which he had met with. None of the patients had been excessively fat, whereas the man in the present case was. In true cases of the syndrome there was marked contrast between the fat distribution in the trunk and extremities, whereas in the present case the distribution was general. Further, Cushing's syndrome was not common in men; and while libido was commonly lost in that condition, it was not so in the present case.

DR. P. W. GRAHAM suggested that the patient's obesity, which followed an acute infection, might be an indication of pathological changes in the hypothalamus.

It was the consensus of opinion among those present that the case was one of cardiac failure associated with obesity.

#### *Asthmatic Tendency Controlled by Cortisone.*

DR. W. T. C. STRAEDE showed a girl, aged seventeen years, who had been first seen in the Asthma Clinic, Alfred Hospital, in April, 1953. She had a history of asthma since seven years of age, with cough, and in the preceding two months had had over 100 injections of adrenaline. The results of investigations including allergy tests were negative. In September, 1953, she was admitted as an in-patient to the Alfred Hospital with asthma, emphysema and left pneumothorax. She responded to bed rest and treatment with aminophylline, adrenaline, potassium iodide and penicillin, and four weeks later her pneumothorax disappeared. She was then treated with autogenous vaccine, and on one day in July, 1954, was in *status asthmaticus*. She attended school for only three weeks in 1953 and not at all in 1954, and her condition remained more or less unchanged until March, 1955, when her asthma became worse and was practically continuous. In June, 1955, her weight was five stone twelve pounds when treatment with cortisone acetate (two 25-milligramme tablets per day) was commenced. There was a dramatic improvement, and in the succeeding month she gained one stone in weight and felt very well, in contrast with her previously miserable existence with repeated injections of adrenaline. As the patient could not continue to receive cortisone indefinitely, Dr. Straede sought advice regarding further treatment.

DR. HAMILTON SMITH considered that the dose of cortisone should be reduced to 12.5 milligrammes eight-hourly rather than 25 milligrammes twelve-hourly.

DR. GEORGE SWINBURNE said that the X-ray report of the patient showed mucosal thickening in the maxillary antra. That might mean anything in an allergic patient, and the girl had no nasal symptoms. He suggested antral lavage; and if the washings were clear, no action should be taken, but if they were purulent, autogenous vaccine might help. If nothing was found on antral lavage, he considered that her outlook was poor.

In answer to a question from Dr. W. J. RAWLINGS concerning the results from injection of placental extracts, Dr. Straede said that opinions he had obtained indicated that they were of no use.

#### *Dissecting Aneurysm of the Aorta.*

DR. STRAEDE also presented details about a man, aged twenty-nine years, who had a history of chorea at eleven years of age and had been accepted for life assurance at twenty-one years. While working on a ladder in January, 1954, he had experienced a sudden onset of severe, crushing, bilateral chest pain radiating to the scapulae, which persisted, and three hours later he was discovered stuporose, cyanosed and dyspnoeic. On examination his heart was found to be enlarged with signs of aortic regurgitation. Blood pressure in the left arm was 170 millimetres of mercury, systolic, and 80 millimetres, diastolic, and in the right arm 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. Lumbar puncture showed no abnormality in the spinal fluid, and the Kline reaction was negative. Radiological examination showed enlargement of the left ventricle, and electrocardiography showed left axis deviation. A provisional diagnosis of dissecting aneurysm of the aorta was made, and twenty-four hours later the patient, who had regained consciousness, complained of constrictive chest pain with numbness and weakness in the left hand. Forty-eight hours after the onset the patient's condition had deteriorated, and he was transferred to the Alfred Hospital. No operation was performed, and the patient gradually recovered; but four months later his chest pain recurred, with loss of consciousness followed by death. The following were the autopsy findings:

Cardio-vascular system: gross left ventricular enlargement with 4-6 oz. straw-coloured slightly purulent fluid in pericardial sac. Dilatation of and loss of intima over first part of aorta with displacement upwards and kinking of the innominate artery with dissection upwards occluding innominate and left carotid artery. Remainder of aorta normal and healthy.

DR. K. NORRIS said that in the type of case under consideration blood was dissecting in the aortic media; if another opening could be made in the intima, blood would return to the aorta and the dissection cease. In the present case operation was not justified, as the patient was improving at a time when operation might have been considered; but dissecting aneurysm was no longer a surgical curiosity and was gradually becoming a treatable condition.



*Carcinoma of the Male Breast.*

Dr. H. C. PURTON presented details of a case of carcinoma of the breast in a male with secondary deposits. He said that male cases constituted 1% of malignant breast lesions. In the present case no secondary deposits were detected in the lungs by X-ray examination, but the patient was dyspnoeic and almost certainly had mediastinal metastases. It was proposed to remove the breast and then treat the patient with deep therapy.

Dr. L. H. BALL said that he had met with three cases of male breast malignant disease in the past two years, in one of which multiple local recurrences had developed three months after operation. Castration of the patient had had no effect, and he considered that in the male breast carcinomata were more malignant than in the female.

*Wound Infection.*

At the evening session Professor Ronald Hare, bacteriologist of St. Thomas's Hospital Medical School, London, gave an address on "Changing Aspects of Wound Infection". At the conclusion there was a "Question and Answer" session. The following are the questions and the answers given by Professor Hare.

Q.: What is the best skin antiseptic?

A.: Iodine is the only one of real value.

Q.: What is your view on wound dressing?

A.: If it is certain that there is no infection in the theatre, use an occlusive dressing—adhesive plaster or a paint. To prevent cross-infection clean patients must not be subject to risk from infected patients, and the latter should be removed to a separate ward with special nurses *et cetera*. The oiling of blankets and floors, wet sweeping, careful dressing technique, attention to nurses' uniforms and the wearing of gloves will all assist in preventing infection.

Q.: Should masks be worn by nurses and doctors on dressing rounds?

A.: There is risk from two microorganisms—staphylococci in the nose and streptococci, which are mainly in the mouth. Streptococci are not expelled by talking, and most mouth organisms are on the tonsils, very few of them being "talked out". Therefore masks are probably of no great value, but doctors and nurses should not touch their noses, should talk softly and should avoid sneezing.

Q.: What is the best skin preparation prior to the application of smaller skin grafts?

A.: The area to be grafted must be cleaned; and while in the early days of penicillin Colebatch found its use adequate, now, with penicillin-resistant staphylococci, it is of no use. No useful advice can be given on the problem.

Q.: Nowadays one sees nurses washing in soap and water and drying their hands on a "clean" towel. Should not sterilization of hands be thorough?

A.: Too much care cannot be given to cleansing the hands; and while repeated sterilization of hands is not always practicable, for example, in the case of a nurse attending to several babies, the use of gloves should be more general, as washing of gloves under a tap results in the rapid disappearance of organisms.

Q.: Of what use is gentian violet for controlling staphylococci on the skin?

A.: It is a good antiseptic and does affect the staphylococci.

Q.: What advice could you give in reference to the treatment of recurrent furunculosis?

A.: Valentine, at London Hospital, found that in most cases the habitat of the responsible organism was the patient's nose, and "Terramycin" or "Aureomycin" ointment used nasally was of help.

Q.: Should streptomycin and/or penicillin be used prophylactically?

A.: The use of an antibiotic "umbrella" is wrong. Antibiotics and sulphonamides should be used only when necessary, not as prophylactics, and then in adequate doses.

Q.: Are there any antagonistic combinations of antibiotics?

A.: In many cases it is preferable to use two antibiotics, but do not use a bactericidal with a bacteriostatic antibiotic.

Q.: Is the development of resistance to antibiotics inevitable or due to their misuse?

A.: Probably both, but development of resistance would have been much less if antibiotics had not been misused.

*Medical Societies.**PÆDIATRIC SOCIETY OF VICTORIA.*

A MEETING of the Paediatric Society of Victoria was held at the Royal Children's Hospital, Carlton, on September 14, 1955. This was the H. Douglas Stephens Memorial Meeting.

*Clefts of the Lip and Palate.*

Dr. A. R. WAKEFIELD gave an address entitled "A Survey of Present Knowledge of Clefts of the Lip and Palate". He said that it was a great privilege to be asked to address the Society at its annual meeting when they paid tribute to the memory of Dr. H. D. Stephens. Because that memory was still fresh in the minds of even the youngest members, it needed no words of his to recall the great influence Dr. Stephens had exerted over paediatrics in Australia, and it was singularly appropriate that on the present occasion the committee should choose a subject which was so near to Dr. Stephens's heart. Dr. Wakefield hoped that he might do justice to it.

Dr. Wakefield commenced by saying that congenital clefts of the lip and palate had been classified in many ways over the years, but from the practical point of view they could be considered in three groups: group I, consisting of clefts of the lip with or without a notch or cleft in the alveolus, which might extend back as far as the incisive foramen; group II, consisting of clefts of the palate alone posterior to the alveolus; group III, consisting of clefts of lip and palate in combination. In all cases there were grades of severity from slight notches to complete clefts, and in groups I and III the clefts might be unilateral or bilateral.

In discussing the embryology of the condition Dr. Wakefield said that for over a century no serious doubt was cast on the "classical" conception of the early differentiation of various processes surrounding the primitive stomodeum, which grew downwards from above and inwards from the sides, finally to fuse, and by their fusion to complete the formation of the nose and lips; and the artists' conceptions of those processes, as they still appeared in the text-books, were well known to all.

However, the work of Pohlmann in 1910 and of Veau in 1936 had failed to confirm the existence at any stage of processes separated by clefts, which had hitherto been accepted without question, and it had been suggested, without going into detail, that congenital clefts of the lip and alveolus, at any rate, developed not from a failure of processes to fuse but from a breaking down of certain areas, where ectoderm and endoderm had remained in contact owing to failure of the normal growth of mesoderm between those two layers. In other words it was a breaking down of an already existing membrane rather than a fusion of processes growing in from the periphery.

In the case of the palate posterior to the incisive foramen, however, it was still believed by most that palatal processes grew together from the side walls of the oral cavity; although the occasional submucous palate clefts, which might be very thin and might break down during life, suggested that even there the sequence of events in cleft palates might be due to a failure of mesodermal growth, leaving ectoderm and endoderm in contact as a thin membrane which subsequently broke down.

There was still a lot of difference of opinion on the embryological concepts mentioned, and it was perhaps sufficient to note that they were not nearly so clear-cut as the old school of embryologists would have had them believe.

As far as the aetiology of the condition was concerned, even as far back as the eighteenth century reports were made of cases of familial incidence of the abnormalities concerned; but although hereditary predisposition was suggested shortly after that it was not until large series of treated patients began to appear towards the end of the nineteenth century that detailed reports of family histories became available in any useful numbers. From that time up to the present many series had been recorded, and in general the more detailed the review the higher had been the figure for familial occurrence of the abnormalities.

All other suggested causes, and they were legion, had been slowly but surely eliminated until at the present day, largely as the result of the exhaustive studies of Fogh-Anderssen, of Denmark, certain facts and a few reasonable assumptions had emerged:

1. Most writers had set the incidence of clefts of the lip and/or palate at about one per 1000 births, more or less.

Fogh-Anderssen's figure for Denmark was one per 665, and it seemed likely that the discrepancy was due more to his more accurate documentation than to a high incidence in Denmark.

2. It seemed certain that they were dealing with two genetically different conditions and that cases of cleft lip alone or cleft lip and cleft palate (in other words, groups I and III) should be considered as essentially the same condition except for a variation in degree, and should be distinguished from cases of isolated cleft palate (group II), which genetically was an entirely different condition.

3. The condition of cleft lip with or without cleft palate occurred predominantly in males and was seen more often on the left side than the right.

4. The condition of isolated cleft palate occurred predominantly in females.

5. A near family history of cleft lip was obtained in about 40% of cases of cleft lip with or without cleft palate.

6. A near family history of cleft palate was obtained in about 20% of cases of cleft palate alone.

7. There was no cross relationship between the two conditions, and the incidence of one condition in families predisposed to the other was no higher than would be expected on the basis of coincidence.

8. Other physical and mental abnormalities occurred in about 10% of the cases. This 10% seemed to occur chiefly in the cases which did not show an hereditary disposition.

9. It seemed likely that at least some of the cases without family history might be due to genetic mutation—after all, the condition had to start somewhere.

10. The method of inheritance was far from fully understood, but in the case of cleft lip with or without cleft palate Fogh-Anderssen's observations indicated a conditioned dominance with sex limitation to males; whereas in isolated cleft palate they suggested single dominance with sex limitation to females. Fogh-Anderssen had first put forward those suggestions in his monograph published in 1942, and in a short paper read at the recent International Congress of Plastic Surgery he had indicated that further observation had served only to confirm those views.

11. From the practical point of view certain other important figures had emerged, which might be of great help in answering the inevitable question which parents asked: "What are the chances of this occurring in subsequent children?" (a) The chance of cleft lip in the offspring when one parent had cleft lip was about 2%—still a very low risk. (b) If one parent had a cleft lip and a child was born with a cleft lip, the risk to subsequent children was about 10% to 15%. (c) If a child with a cleft lip was born to two normal parents, the risk to subsequent children was about 5%. (d) If there were other cases of isolated cleft palate known in a family and one parent had the condition, the risk to the children might be as high as 20%, particularly if there was already one child with the condition.

Dr. Wakefield said that he had laid considerable stress on the question of etiology and on Fogh-Anderssen's investigations, because he found that, outside those few surgeons who had a particular interest in the subject, there was very little general knowledge of the condition, and most people still seemed content to regard the conditions as acts of God which appeared without rhyme or reason.

There was still a lot to discover about the conditions which determined manifestation of the abnormalities, whose rate of manifestation was only about 50%, but there was no doubting the fact that heredity was by far the most important factor in determining the incidence of the condition.

In discussing management, Dr. Wakefield considered the problems of feeding, the time for operation, and the type of operative treatment.

#### Feeding.

In discussing the early feeding of the affected babies, Dr. Wakefield said that it was necessary to refer back to the three clinical groups. Group I (clefts of lip only) could be dismissed very quickly by saying that the children could be fed in exactly the same way as a normal child, and under no circumstances should some special feeding method be introduced for the sole reason that the child had a cleft lip. Those children had intact palates, and they could both suck and swallow without difficulty.

The feeding of children in groups II and III could be considered together. In both cases the palate was cleft, and the additional lip cleft in group III made little difference from that point of view. The children could not suck

effectively, but they could swallow. It followed that all that had to be done was to place the milk over the back of the tongue, preferably with a spoon; the child, in the absence of any other abnormal state, was fully capable of doing the rest, and did.

Why then was there so much trouble about feeding those babies? Dr. Wakefield was quite sure that most of the troubles were due to two common misconceptions. The first was that because the child might get an ounce or so of fluid in by a great deal of effort at inefficient suction, it was worth while persevering with suction methods in the hope that the mechanism might become more efficient. In fact, the child struggled harder and harder for less and less; and by the time the effort was abandoned, he had become a "feeding problem" and a "failure to thrive". An intelligent mother would often solve the problem quicker than the doctor by enlarging the hole in the teat, so that the milk could be poured over the tongue and did not have to be sucked out.

Dr. Wakefield said that it remained a mystery to him why some paediatricians persisted with such clumsy and inefficient compromises as dental appliances when spoon feeding could be established from the very first feeding and the mother shown how to do it from the first day; so that neither she nor the child need remain in hospital any longer than if the child was normal. He said that he was completely unimpressed by talk of the psychological importance of suction in relation to those children. If a half-empty stomach, a chronic sense of hunger, an inefficient suction mechanism and an uncomfortable bung in the mouth were compared with a sense of gastric satisfaction achieved with a minimum of unnecessary effort, he had a fair idea which child would be better adjusted to its environment.

The other and much more important misconception that existed was that because a child had a cleft palate it could not swallow properly and needed to be fed by gavage. In fact, nothing was further from the truth, and it could be laid down with great emphasis that cleft palate *per se* was never an indication for gavage feeding. A child with a cleft palate could not suck adequately, but could swallow adequately; if that simple fact was more generally appreciated, not only by practitioners, obstetricians and paediatricians, but also by sisters-in-charge of nurseries, many an otherwise normal child would be saved from the downhill path that followed the indiscriminate and often unskilled use of gavage feeding in those children.

There were three groups of babies with cleft palates which failed to come up for operation in a satisfactory general state at the proper time. The first group consisted of the 10% who had associated abnormalities, birth injuries or neonatal illnesses, or who were born prematurely. In those cases all sorts of difficulties were encountered, and gavage feeding had commonly to be used—but not because the children had cleft palates. They would always be a problem; a proportion would die, and many would be chronic invalids. The other two groups comprised otherwise normal children, and the difficulties were man-made, either by attempting to establish suction feeding or by unnecessary and often unskilled gavage feeding, with all its attendant complications.

Dr. Wakefield said that he had stressed that aspect of the management of the babies because he was afraid that paediatricians must carry some of the responsibility for the state of general ignorance that had existed on the subject. In the vast majority of cases the mother could be shown how to feed the child with a spoon from the very first feeding; yet time and time again the mother never got near the baby while sisters and doctors experimented with various ineffective methods involving suction or passed straight to intragastric feeding. He had on many occasions been asked to see these babies in hospital within a week or two of birth when the only reason for their admission to hospital was that so far nobody had bothered to try the effect of pouring a spoonful of milk over the back of the tongue, and it was automatically assumed that the child would become a "feeding problem", in the solution of which the mother could have no possible part.

Fortunately that sort of thing was becoming less frequent, but it still accounted for far too many of the puny, ill-nourished, wheezing wretches of babies that failed to reach the fairly rigid standards set for operation at the optimum time.

#### The Times for Operative Treatment.

Dr. Wakefield went on to say that if one assumed that the baby was thriving, the best time for operative repair of the defects had now to be decided.



In group I, the group of pre-alveolar lip clefts, there was usually little or no skeletal abnormality, and the nasal deformity was not gross. There was no urgency about the repair, and personally Dr. Wakefield thought that he could deal with them better when the structures were bigger than they were in the first few weeks. Anaesthetic risks decreased as the child grew older, and furthermore the children were frequently fed at the breast, and it seemed unnecessary to interrupt that to perform an operation that was purely elective. He therefore repaired those clefts at somewhere between six and nine months as a general rule.

In group III, the group of complete clefts, there was usually a fairly gross distortion of soft-tissue structures and a skeletal deformity, particularly in the bilateral clefts, which seemed to grow worse and more difficult to repair as the months went by. The deformity was a hideous one to the lay mind, and from all points of view, therefore, closure of the lip and restoration of the nasal contour were desirable as early as possible. Surgeons varied a little in what they regarded as the optimum time for the lip repair in these cases, but most operated at two to three months. Most of them, he thought, liked to have a baby somewhere about ten pounds in weight and thriving. The palate element of these clefts was treated along the same lines as in the group II cases.

In group II, and also in group III, after the lip was repaired the choice of time for palate repair was governed by a number of factors. The technical difficulties of operating in a confined space, the severity of the procedure, the early anaesthetic risks, and the substantial development of the palatal processes which occurred during the first year were all factors which postponed operation for as long as possible; whereas the desire to have the palate repaired and mobile by the time true speech was commencing made the surgeon want to do the operation well before that time. Dr. Wakefield considered that the best compromise was to select a time at about twelve months of age.

In that regard, however, he thought that they must consider seriously the case for later operation that had been brought forward in recent years by those whose main concern was the child's dentition. Those people had maintained that if the palate repair was deferred for about five years, interference to the alveolar arch and the developing dentition during its most active phase of development would be avoided, and that such a delay in operation was not prejudicial to speech development. He could not agree with that point of view, which he thought was based on a number of false premises.

The first wrong assumption was that surgical palate repair did in fact interfere with the development of the alveolus and the contained dentition. That might have been so when Brophy devised the vicious procedure which bore his name, but there was not a jot of evidence that the modern operations which were confined to soft tissues had any such effect.

The second, incorrect assumption was that delay in operating for several years did not prejudice speech production. Dr. Wakefield had no doubt at all that such a delay was gravely prejudicial to the best speech results that could be obtained, and he was emphatic that the production of the best possible speech was so far transcending in importance any other feature of the management of the children that, even if early operation did disturb the developing dentition to some degree, he would accept this as a reasonable price to pay for the better speech that would be obtained thereby.

There were three aims in the treatment of these clefts: (i) good speech, (ii) the best possible external appearance and (iii) a good dentition in reasonable occlusion; whilst the third must be respected at all times during the surgical procedures necessary for the first two, it would always remain the third in importance when one was considering priorities in the overall management of the patients.

Apart from the primary operations for lip and palate, certain secondary plastic operations were frequently necessary on the lip and nose, particularly in bilateral cases. The best time for those was probably about four years of age, or just before the child started school.

#### Operative Treatment.

Dr. Wakefield said that it had been his object in his paper to provide a fairly general review of the subject and to consider chiefly the principles which must guide management.

In considering operations on the lip and nose the basic principles were simple: (i) to provide a depth of lip from nasal floor to vermilion edge equal to that of the opposite side, at the same time retaining the maximum side-to-side length of muco-cutaneous line; (ii) to free the old nasal at

its base and by incision up the lateral nasal wall sufficiently to allow of a near-normal nasal contour after repair of the nasal floor and lip; (iii) to carry the repair of the nasal floor sufficiently far back at the first operation to obviate the risk of post-alveolar holes following on the later repair of the main palate.

Very few indeed of the innumerable operations that had been devised respected those principles, and most of the earlier procedures had now been discarded in favour of those few. In unilateral cases the procedures described by Brown and McDowell, of America, and by Lemesurier, of Canada, with various modifications were probably those most widely used today, and both respected the principles described. In bilateral cases they did not believe that the first principle could be satisfied if the whole of the mucocutaneous line on the prolabium was sacrificed, as in most of the methods advocated at the present day, and Dr. Wakefield endeavoured to use as much as possible of that in his repairs. Only by so doing could one obtain a lip which was long enough from side to side, and avoid the necessity for an Abbe type of repair later in life.

In regard to palate repairs the following were the principles of treatment:

1. To obtain a palate of sufficient mobility and sufficient length from before backwards to allow the palate to act in conjunction with a contracting pharyngeal musculature to produce a competent naso-pharyngeal sphincter. It must be remembered that the part of the soft palate which made contact with the pharyngeal wall was not the back edge, but the upper surface about one-third of its length in front of that, and the significant relationship was between the length of the palate back to that point on the one hand and the distance from that point on the palate to the posterior pharyngeal wall on the other hand. The size of the pharynx varied greatly in different children, and a length of palate that was adequate in one child might not suffice in another. The bulk of adenoid tissue present might also influence the efficiency of the sphincter mechanism, and an ill-advised operation for tonsils and adenoids in these children might sometimes make the difference between fairly good and bad speech.

2. To obtain a palate which was intact in itself and had no holes through which air might escape.

Dr. Wakefield said that a number of operations had been devised based on those principles, but in general he felt that an adequate effective length of palate was obtained only if the front ends of the dissected palate flaps were detached and the flaps allowed to fall backwards as a whole, as in the procedures attributed to Veau, Kilner and Wardill, which were similar in type, and that of Dorrance, in which the principle was the same but the execution different. The older Langenbeck procedure rarely achieved the necessary length of palate, and Dennis Browne's modification of it seemed to Dr. Wakefield to be based on an entirely wrong conception of the mechanism of the palate.

#### The Late Management.

The later management after all operative treatment was completed resolved itself into an assessment of speech and any other factors which might influence it, such as mental development and hearing ability about the age of three years, and the commencement of speech training if indicated at that age, together with periodic dental examination and orthodontic treatment or prosthetic replacement as necessary.

Dr. Wakefield hoped that in his somewhat sketchy review he had been able to convey some sense of the magnitude of the overall problem presented by the children considered and of the necessity for an orderly plan of treatment by a team of experts over a period of years. It was only by such organized teamwork that they could avoid that unhappy fragmentation of treatment which resulted so often in innumerable ineffective operations with inadequate attention to speech training and orthodontia, and often complete disregard for the child's personality. It should be the aim to complete the surgery in as few operations as possible, and in any case to have the operative programme finished by the time the child started school. He believed that that could be done in all cases; for in the final analysis it was the quality of the primary operations on the lip and palate which determined the results, and no amount of secondary surgery on either the lip or the palate could ever make up for incompetent primary treatment.

#### Discussion.

Dr. J. COLEBATCH asked Dr. Wakefield whether he knew anything of the possible relationship of animal experimental

work to the human disease, and whether that threw any light on the etiology of the condition. Dr. Colebatch said it had been shown in experimental animals that cortisone would produce cleft palate or some type of facial cleft in a high proportion of cases if given at a certain stage of pregnancy, and it had also been shown that the incidence under those circumstances was greater in some strains of animals and in some circumstances than in others. It would therefore seem that, in animals anyway, other factors as well as the genetic one were important. Dr. Wakefield had said that in 10% of cases there were associated abnormalities; Dr. Colebatch wondered whether it was known if there were any other factors associated with the development of the defects in those cases.

Dr. Wakefield said, in reply, that with regard to the 10% of cases in which there were associated abnormalities, they seemed to fall in the 60% in which there were no family histories of the condition, and then, of course, there must be other factors responsible. Andersen's idea of conditioned dominance was interesting, but what were the conditions? There was only about 50% genetic manifestation of the defect, and there must be other factors that determined whether it was going to be manifest or not. It might be that the genetic make-up as a whole was important. That might influence whether the particular gene became dominant. With regard to animal experiments, a certain amount of work had been done on trying to establish families with these abnormalities, but it had not reached the stage where anything absolute had come out of it, except that the family histories of the animals had tended to confirm Andersen's observations in humans. Many other facets of the problem had been mentioned; for instance, in micrognathism the theory for the cleft in the palate was a mechanical one, in that the palate could not fuse because of the smallness of all structures in the floor of the mouth. Also it was well documented that some children had been born with amniotic bands through those clefts. There was probably not sufficient information to incriminate any of the factors indisputably. However, the wealth of clinical material in the Royal Children's Hospital would offer an opportunity for study.

Dr. Colebatch also asked how far the families were investigated, and what constituted a near-family relationship.

Dr. Wakefield said that that was of significance. Some of the early investigators had drawn all sorts of conclusions from the fact that relatives as far back as seventh cousins had the abnormalities. It would be found that 27,000 individuals lay between those two relatives, and at about the rate of one per thousand births one could expect 27 cases. So it had to be a near family relative, but he could not say how far one must go.

Dr. W. FORSTER said that he had found it was often hard to change transverse anterior nares into antero-posterior nares; he wondered whether Dr. Wakefield meant, when he said that only one operation should be necessary for a child with a hare-lip, that he attempted to alter the shape of the nares at the first operation.

Dr. Wakefield said that one could achieve a good result in one operation in cases of prealveolar clefts when there was no substantial skeletal abnormality and the degree of nasal distortion was small. Even when there was minor distortion, he endeavoured to correct it the first time, because if he could not do it then, he was unlikely to achieve anything better the next time. It was a different matter in the group III cases, in which there was a cleft lip and palate with great distortion.

Dr. M. CLARKE asked two questions: firstly, whether Dr. Wakefield had ever come across a palate which was congenitally so short that he was unable by any technical means to lengthen the palate sufficiently to make speech reasonably good, and whether he had used Wardell's pharyngoplasty operation in that respect; secondly, whether Dr. Wakefield took any steps before the child reached the age of three to instruct the mother in speech, and in follow up how often he saw the palate subsequent to the commencement of speech therapy.

Dr. Wakefield answered the second question by saying that any organized form of speech therapy was difficult, and the development of speech was hard to assess prior to three years of age. By and large he thought that that was a practical time to start. One frequently had a talk with parents before that and told them a few helpful points. During follow up he saw them with the speech therapist for a start, and then decided, rightly or wrongly, whether the child and the parents should be referred to the psychiatrist. He thought that the larger number of them did not need to harass the psychiatrist at that stage. He then saw them subsequently at the request of the speech therapist.

She might feel sometimes that the palate was not long enough, and asked if anything could be done. That brought Dr. Wakefield to Dr. Clarke's first question; he said that one saw a short palate particularly in association with congenital absence of the vomerine part of the nasal septum. In that condition there was a bunched-up palate anteriorly and very little substance at all in the palate posteriorly. Nearly all the children had a rather flattened nose and very little in the way of nasal septum. He did not know how one repaired those. One could obtain a side-to-side closure, but never one adequate in length. He had the impression that that condition occurred more commonly in relation to mental deficiency than other cleft palates. There were other cases in which the palate seemed of reasonable length, but there was a much larger pharynx than normal. He thought that a better operation than the Wardell operation was that of Heinz, of Sheffield. That seemed to be a useful type of pharyngoplasty, in which two rather long flaps from the Eustachian tube area downwards were dissected out, the underlying mucosa was closed directly, and the flaps were transposed upwards, joined across the mid-line and set in to a transverse incision. That gave a much bigger and more bulky ridge onto which the soft palate might close. He had not so far used that technique, but many surgeons he had spoken to were impressed with the operation for that type of case.

## Out of the Past.

*In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.*

### HEALTH OF THE CONVICTS.<sup>1</sup>

[From "An Historical Journal of the Transactions at Port Jackson and Norfolk Island", by John Hunter, Esq., Port Captain in His Majesty's Navy, London, 1793.]

NOVEMBER, 1791: From the debilitated state in which many of the convicts were landed from the last ships the number of sick were greatly increased; the surgeon's return being upwards of 400 sick at Parramatta; and the same day medicines were distributed to one hundred and ninety-two at Sydney. To the number of sick at Parramatta upwards of one hundred may be added who were so sick they could not be put to any kind of labour nor even to that of pulling grass for thatching the huts. Forty-two convicts died in the month of November, and in these people nature seemed fairly to be worn out: many of them were so thoroughly exhausted that they expired without a groan, and apparently without any kind of pain.

## Correspondence.

### THE BAN ON HEROIN.

SIR: I am deeply concerned at the ban on heroin and the manner in which it has been imposed. I think it is time the profession took some concerted action in the matter. This is a ban which has been imposed at the instance of politicians, and it should be strongly resisted.

In August, 1952, the Federal Council considered this matter at the request of the Commonwealth Government. It decided that there should be no curtailment on the availability of heroin to the medical profession.

On a further request from the Government for careful and sympathetic consideration of the matter the Federal Council in February, 1953, after receiving advice from five State Branch Councils that they were not opposed to the ban, reversed its previous decision and resolved to support the ban. The responsibility then rests squarely on the Branch Councils.

So far as I can ascertain members of State Branches were not asked for their opinions or given any opportunity to

<sup>1</sup> From the original in the Mitchell Library, Sydney.



express them. I feel that this was a serious omission and should be remedied forthwith.

It would be quite easy for State Councils to send out a simple *questionnaire* to their members on these lines: (1) Are you in favour of a complete ban on the use of heroin in Australia? (2) How many cases of heroin addiction have you seen in the past ten years?

If a majority favours a ban, then there is little more to be said. If it does not favour a ban, then the Federal Council should take further action.

The Federal Minister for Health has stated that if the Federal Council believed that the ban should be lifted, the Government would seriously consider doing so.

This may be our last chance. It is up to the profession as a whole to make its wishes known. I suggest that members request their Branch Councils to send out a *questionnaire*. When the replies are received and collated, then, and then only, Branch Councils will be in a proper position to further advise the Federal Council.

Yours, etc.,

D. R. W. COWAN.

163 North Terrace,  
Adelaide,  
January 5, 1956.

SIR: The recent ban on heroin in New South Wales is awakening the medical profession to give expression to its disapproval. A number of practising doctors have pointed out that there is no adequate substitute for heroin in many ailments: in certain forms of malignancy, prolonged childbirth, advanced tuberculosis, and for all patients in severe pain from whatever cause who react adversely to morphine.

One of the most eloquent, though silent, arguments in England against the ban on heroin was the accumulation of large stocks of heroin by most, if not all, of the large hospitals in London in anticipation of the government ban which was to have operated as from December 31 last.

An interesting development is the comparatively recent incidence of addiction to pethidine in countries where there was neither ban nor heroin addiction—pethidine, one of the very drugs the pharmacologists say is an adequate substitute for heroin!

The legislation in the United States of America against heroin, which has been in existence for twenty years and upwards, withheld it from its legitimate use by doctors for the alleviation of suffering; but it is in the same America that heroin addiction is at the moment a problem. This shows that legislation against this drug does not in itself stop addiction: indeed, it probably adds to it, since it creates the fields in which the "black marketeers" till, sow and, in due course, reap their harvests.

Legislation against the open and lawful use of alcohol in America created one of the greatest civic monsters of modern times, namely, the "moonshine" gangster, who, at one period, was a thousand times more dangerous than any goblin which alcohol ever brought into being. Similarly, prohibition of the legitimate manufacture and use of heroin will probably have little, if any, effect on the incidence of addiction in any country, for heroin is quite easily manufactured from either morphine or crude opium, which are produced yearly in hundreds of tons—tons, not pounds.

After all, there was really no need for a ban here, for there is no addiction, and, virtually, all doctors are fully conscious of their responsibility in this matter and of the sanctity of the human lives that constantly come under their care. The fault of this legislation lies, perhaps, not so much with the New South Wales Government as with the advisers whom, it is presumed, the Minister consulted. It is to be hoped that representations will be made to the Government to reconsider the question as the British Government has done, so that further light may be directed upon this subject, which is of such moment in the realm of human suffering.

Yours, etc.,

WALTER J. HULL.

Burwood,  
New South Wales,  
January 9, 1956.

#### OBSERVATIONS UPON 250 CASES OF BLEEDING PEPTIC ULCER.

SIR: I desire to reply to some of the comments on my "Observations Upon 250 Cases of Bleeding Peptic Ulcer" which have appeared in several issues of your journal since the article was published on November 12, 1955; particularly because I feel that some of these comments are based upon an insufficiently careful reading of the article.

One of the main problems discussed in the article was the problem of the selection of cases of bleeding ulcer in which surgical treatment offered the only hope of preventing a fatal outcome. The complaint was made that the criteria or formulae commonly advocated to select these patients were platitudinous and vague, were very difficult to apply to actual cases and, when applied, were very inefficient in separating the proper cases for selective surgery. This complaint was supported by applying in retrospect some of the commonly advocated formulae to a series of 250 carefully documented cases of bleeding ulcer, which had been treated almost entirely by medical means; by so doing it was demonstrated that these formulae (chiefly concerned with recurrent or continuous bleeding), even if they could have been applied to this series of cases, would have fallen very far short of their objective of separating the cases in which surgery was obligatory, that is, the fatal cases.

The conclusion was then suggested that unless some more efficient formula for indicating selective surgery was forthcoming, the best results might be obtained "by treating all patients with bleeding peptic ulcer non-surgically and accepting a certain mortality". Dr. Brosnan and Dr. Newton in their letters disagreed with this conclusion and affirmed that surgery should have a place in the treatment of bleeding peptic ulcer; but in regard to the all-important question of the method of selection of cases for surgery, all they did was to restate several of the very platitudinous formulae with which I had dealt, and completely ignored my criticisms of them. This tactic in debate is, of course, very difficult to combat.

However, I think that the formulae for selective surgery put forward by Dr. Brosnan and Dr. Newton are worth considering in some detail, for they are excellent examples of the platitudinous statement which I criticized in my article. Dr. Brosnan says that the golden rule is "to detect early those cases which fail to stop bleeding and to check this bleeding by surgery". Dr. Newton says "surgery should be seriously considered in patients over the age of forty years whose initial haemorrhage continues despite treatment in hospital or who have recurrence of bleeding after admission".

I suggest that these statements (and others like them) are quite meaningless and quite useless as a practical guide to selective surgery, and quite incapable of application to the average case of bleeding ulcer; they denote lack of familiarity with the natural history and behaviour of this lesion. At precisely what point does one decide that a given case has "failed to stop bleeding" and when is it "early" in such a case? What is the meaning of the phrase "surgery should be seriously considered . . ." Does Dr. Newton mean that all cases of the type he goes on to specify should be operated upon, or only some of them? If only some of them, which ones? At what stage in terms of hours or minutes is it decided that the "initial haemorrhage continues despite treatment"? Has not the rate of blood loss any bearing on this decision? Is "recurrence of bleeding after admission" to be considered an absolute indication for surgery irrespective of the amount of blood lost? What if the patient vomits half an ounce of blood; what if the recurrent bleeding (as often happens) does not enter the stomach but passes through the bowel and is not evident till some hours or days later? The difficulties of estimating when bleeding starts and stops, and how much blood is being lost, cannot be overstated.

Many of these phrases appear to be based on the conception of a bleeding ulcer behaving like a running water-tap and continuing to bleed at a steady rate until it is "turned off". Actually, of course, the bleeding from an ulcer is generally intermittent, with unpredictable variation in the rate of bleeding and in the intervals between bleeds. Careful history-taking will reveal that a large percentage of patients have been bleeding (*per rectum*) intermittently for days or weeks prior to admission. Once the bleeding reaches a certain rate the patient is doomed, and this rate may develop at the outset, or it may suddenly supervene at any time later when the patient is apparently doing well; in either case the patient is dead within an hour or so, and no opportunity exists for surgical treatment. By the time it has become apparent to even the most astute observer that medical treatment has failed, it is already too late for surgical treatment. It is at this stage that the surgeon, seeing the case for the first time, is apt to feel that the physician has waited too long before calling him.

If there is a place for surgery in the treatment of bleeding peptic ulcer, then it should be possible to state the indications for its use in precise and exact terms; it is also necessary to demonstrate that the overall mortality and morbidity resulting from the use of any formula for selective surgery is less than that resulting from purely medical treatment,

bearing in mind that the mortality rate in partial gastrectomy done as a true emergency to arrest bleeding is probably at least 10%.

Dr. Newton claims that in my reported series 16 cases died of recurrent bleeding out of a total of 69 patients with this condition; I make it nine out of 63—I would not include the six cases dying after partial gastrectomy in such an estimate in this context. Nevertheless, there is no argument on the general proposition that mortality is highest in such cases; the point at issue is whether and precisely how this mortality can be reduced by selective surgery.

Dr. Newton quotes me as saying I was "content to accept an inevitable mortality"; actually I said no such thing, and the word "content" does not appear in the article. I said that in the absence of a satisfactory formula for selective surgery the inevitable mortality associated with medical treatment might be the best that could be achieved—which is a very different thing; and nobody who read the article with any care could possibly infer, as Dr. Brosnan has done, that I regarded that inevitable mortality as being in the region of 10%. Certainly the overall mortality of the reported series was around 9%, but it was apparent that many of the cases in this series had not been treated according to the medical regime finally adopted and advocated in the article; indeed six of the twenty-two fatalities followed surgical treatment.

Dr. Brosnan regards the use of an indwelling Ryle's tube very highly as a means of obtaining information about recurrent or continuous bleeding. I have found that the information to be gained in this way is not sufficiently reliable or important to offset the fact that it interferes greatly with several important aspects of medical treatment. A great deal of slow bleeding from duodenal ulcers never enters the stomach, and brisk bleeding can be detected without a Ryle's tube.

Dr. Newton characterizes my advocacy of restricted transfusion (as opposed to free transfusion) as "the height of foolishness", as "courting disaster" and as "putting the clock back twenty years". Though it was admitted that in regard to the particular series of cases published no statistically valid proof could be advanced for my conclusions in regard to blood transfusion, nevertheless, these conclusions are based upon an extensive experience of both methods in some hundreds of cases.

Dr. Newton does not say upon what experience his contrary opinion is based; this fact taken in conjunction with the rather intemperate tone of his criticism (to say the least of it) leads one to believe that his opinion is based upon nothing more than a sense of outraged orthodoxy, an attitude which has never contributed much to the advancement of medicine. The fact that there is no statistical proof in the literature for my statement that free transfusions tended to cause recurrence of bleeding by no means disproves the statement, and to the best of my knowledge the proposition (in relation to peptic ulcer) has never been thoroughly investigated on a statistical basis.

I should like to say that in the very small number of cases treated surgically in this reported series several surgeons, all of first-rate ability and repute, were concerned. The number treated surgically was obviously so small as to be statistically without significance. However, the recorded fatalities do point up a fact which is generally admitted, namely, that a considerable mortality rate is associated with partial gastrectomy done as a true emergency to arrest bleeding, and this mortality rate becomes increasingly significant as the indications for operation are widened.

Yours, etc.,

Repatriation General Hospital,  
Concord,  
New South Wales,  
January 10, 1956.

W. K. MANNING.

#### ADVERTISEMENTS AND SAMPLES FROM DRUG HOUSES.

SM: Heartened by Dr. W. Alexander Dunn's belated, but philosophical, understanding, may I offer a few comments on the distribution of advertisements and samples to the medical profession?

The "great nuisance" of disposing of advertisements and not too bulky samples (Leo Loorham, M. J. AUSTRALIA, September 24, 1955) can be solved by a simple filing system—a box of nine inch by six inch envelopes, a secretary to list contents, indications *et cetera* on the envelopes, and a suitable

drawer or box for filing. Obsolete items should be discarded at suitable intervals. If used in conjunction with the "Physician's Index" (Butterworth and Company) much of the indexing can be eliminated and the value of the filing system enhanced.

Patients are usually delighted with a free gift from the doctor, and, used judiciously, samples increase goodwill between doctor and patient. More important, of course, both are often most grateful for the therapeutic results.

I am sure all drug houses would like their representatives to make more personal calls on doctors—especially if these are preferred—instead of depending to a large extent on circulars. But there is a shortage of suitable men, and the cost of £1 to £2 for travellers' calls often makes them prohibitive.

Complaints of samples of poisons—not having been requested—received in the post (Campbell Duncan, M. J. AUSTRALIA, November 5, 1955, and "Medical Practitioner", M. J. AUSTRALIA, November 19, 1955) might well be referred to the trade associations. One organization I know of has asked its members not to post such samples, except on request.

With Dr. Dunn's letter (M. J. AUSTRALIA, December 31, 1955) I most heartily concur.

Yours, etc.,

E. H. FLORANCE,  
Representative of Silten Limited,  
Hatfield, Herts, England.

9 Canrobart Street,  
Mosman,  
New South Wales.  
January 5, 1956.

## Obituary.

ROY BALDWIN MINNETT.

THE following appreciation of the late Dr. Roy Baldwin Minnett has been prepared by a colleague who wishes to remain anonymous.

On June 13, 1886, in Willoughby Street, Milson's Point, New South Wales, there was born to Mr. and Mrs. J. A. Minnett a son who received the names of Roy Baldwin. His father was manager of the New Zealand Insurance Company for many years, and his mother's maiden name was Milson. She was a relative of the Milson family after whom Milson's Point is named. For a period of nearly seventy years Roy was to be a treasured friend to many and a delight to multitudes who saw in him the personification of the Ideal Australian sportsman.

From 1900 to 1906 he was a pupil of the Sydney Church of England Grammar School, on the North Shore, where his academic and physical prowess made for him a permanent name as a great son of a great school. In athletics and in cricket he gained the highest school honours—champion athlete, captain of the first eleven, cricketer colours for four successive years, representation in the Combined Great Public Schools eleven for three years, centuries galore, hat tricks *et cetera*—seldom has one youth attained so much glory in the outdoor. What of the academic side? Steady progress with a pass in the Junior Examination and finally matriculation for the Faculty of Medicine showed his scholastic ability.

A bare recital of his many achievements at school gives no measure of the character of the individual, but modesty and an unassuming nature plus a friendliness to all others made a character so outstanding that it comes as no surprise to find that he was also Senior Prefect at "Shore". His only son was to occupy the same position a generation later.

Roy Minnett entered the medical school of the University of Sydney in 1909 and graduated in 1915. During his course he mixed sport and work in the same measure that he had at school; although his course was interrupted by representation in Sheffield Shield and in test matches, as well as by the supreme cricketer honour of membership of the touring Australian cricket team of 1912, he still found time to enter the credit list of his fourth year examinations and to graduate comfortably.

The university years were his great years in representative sport, and he gained "blues" for cricket and baseball. Among the many highlights of his cricketer career were the following. He obtained the highest Australian score in the fourth test at Melbourne in 1912 (56 runs). Incidentally in



that test Roy bowled Rhodes after the latter had scored 179 runs in an opening partnership of 323 runs along with Jack Hobbs; this is still the record. On another occasion in a Sheffield Shield match Roy scored 216 not out. In the six test matches in which Roy played he averaged 25.75 runs and took 26 wickets for an average of 20—sure statistical evidence of a great allrounder in our most traditional sport.

After entry into general practice he played with and captained Manly Cricket Club for a number of years and was thus able to continue to take an active part in the sport he loved so well.

It is of interest to note that Roy was not the only one of his family to gain representative honours at sport. His two brothers, Rupert Minnett and the late Leslie Minnett, also played interstate cricket and baseball.

In April, 1917, he married Miss Doris Sinclair Morton, whose sister was married to his brother Leslie.

I am indebted to Dr. Harold Thomas who takes up the thread at this point and writes as follows:

In 1915 Roy and I journeyed together to England, joined the R.A.M.C. and were posted to a Field Ambulance Training Depot at Eastbourne. After three months Roy was detailed for duty in France. On his return to Australia in 1917 he accepted an offer from my father, Dr. David Thomas, that he should join my father and myself in partnership in Manly.

Roy's happy personality and keenness in his work soon made him a very busy practitioner. His skill in midwifery brought him into constant demand for confinements—the majority of these cases in the early days were in the patient's own home—this added considerably to his work. Roy's cheerful demeanour, his generosity, his habitual kindness, his unassuming modesty, were all facets of a very pleasing character.

For many years he was an honorary physician at the Manly District Hospital, and amongst various other honorary appointments he was honorary medical officer to the Baby Clinic at Manly—there as elsewhere his experience and advice were held in very high regard.

The loss of his only son, Peter, when a prisoner of war, came as a very great shock to Roy, and only those closely associated with him could realize how great this was.

In 1951 we decided to retire together. For thirty-five years I had had the privilege of working with him, and, as the years went by, of appreciating more and more his qualities. Those that knew him best will miss him most.

On leaving general practice Roy spent some time as medical officer on coastal vessels and then took an appointment with the Australian Mutual Provident Society. A few months before his death he purchased a small property at Bringelly, New South Wales, and it was while putting his boundless energy and enthusiasm into this new venture that he had a cerebral haemorrhage. On October 21, 1955, he died.

Those of us who had the pleasure of working and playing with Roy knew of the great kindness of his nature and of the everlasting energy he put into helping other people. His interest in Rotary was one expression of that desire to be of service. The unfortunate, the unhappy and the erring were all supported and helped by Roy's understanding and aid.

In his own home Roy and his wife, Doris, offered the most generous hospitality, and rarely did an evening pass that someone did not enjoy their company at short or no notice.

At his funeral service was the unsolicited presence of so many friends that the congregation included knights and pensioners, sportsmen and scholars, labourers and members of the professions. There were mothers and their daughters whom he had delivered to them and whom he had later attended when these same daughters gave birth to their children. The church could not hold the multitude, and the famous Manly Corso was packed on both sides with silent people paying homage to their departed friend.

Roy is survived by his wife, Doris, and his two daughters, Mrs. Nugent Wallman, of Neutral Bay, and Mrs. Robert Lang, of Tamworth. His son, Peter, as recorded earlier, died as a prisoner of war in Japanese hands. His

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 24, 1955.<sup>1</sup>

| Disease.                                   | New South Wales. | Victoria. | Queensland. | South Australia. | Western Australia. | Tasmania. <sup>a</sup> | Northern Territory. | Australian Capital Territory. | Australia. <sup>a</sup> |
|--|------------------|-----------|-------------|------------------|--------------------|------------------------|---------------------|-------------------------------|-------------------------|
| Acute Rheumatism .. ..                     | 2(1)             | 4(3)      | 4(2)        | ..               | 1                  | ..                     | 1                   | ..                            | 12                      |
| Amoebiasis .. ..                           | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Ancylostomiasis .. ..                      | ..               | ..        | 6(1)        | ..               | ..                 | ..                     | ..                  | ..                            | 6                       |
| Anthrax .. ..                              | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Bilharziasis .. ..                         | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Brucellosis .. ..                          | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Cholera .. ..                              | ..               | ..        | ..          | ..               | 1                  | ..                     | ..                  | ..                            | 1                       |
| Chorea (St. Vitus) .. ..                   | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Dengue .. ..                               | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Diarrhoea (Infantile) .. ..                | 6(6)             | 14(13)    | 7(6)        | ..               | ..                 | ..                     | ..                  | ..                            | 27                      |
| Diphtheria .. ..                           | ..               | 4(1)      | ..          | ..               | 8(7)               | ..                     | ..                  | ..                            | 12                      |
| Dysentery (Bacillary) .. ..                | ..               | ..        | 6(6)        | 1                | ..                 | ..                     | ..                  | ..                            | 7                       |
| Encephalitis .. ..                         | ..               | 1         | ..          | 1(1)             | ..                 | ..                     | ..                  | ..                            | 2                       |
| Filariasis .. ..                           | ..               | ..        | 1           | ..               | ..                 | ..                     | ..                  | ..                            | 1                       |
| Homologous Serum Jaundice .. ..            | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Hydatid .. ..                              | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Infective Hepatitis .. ..                  | 62(18)           | 85(42)    | ..          | 16(5)            | 8(3)               | ..                     | 2                   | ..                            | 173                     |
| Lead Poisoning .. ..                       | ..               | ..        | ..          | ..               | 1                  | ..                     | ..                  | ..                            | 1                       |
| Leprosy .. ..                              | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Leptospirosis .. ..                        | ..               | ..        | 9(1)        | ..               | ..                 | ..                     | ..                  | ..                            | 9                       |
| Malaria .. ..                              | ..               | 1(1)      | ..          | ..               | ..                 | ..                     | 1                   | ..                            | 2                       |
| Meningococcal Infection .. ..              | 1                | 6(6)      | 1           | ..               | ..                 | ..                     | ..                  | ..                            | 8                       |
| Ophthalmia .. ..                           | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Ornithosis .. ..                           | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Paratyphoid .. ..                          | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Plague .. ..                               | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Pollomyelitis .. ..                        | 4(1)             | 4(4)      | 1           | 6(3)             | ..                 | ..                     | ..                  | ..                            | 15                      |
| Puerperal Fever .. ..                      | ..               | ..        | 1           | ..               | ..                 | ..                     | ..                  | ..                            | 1                       |
| Rubella .. ..                              | ..               | 104(82)   | ..          | 6(4)             | 3(3)               | ..                     | ..                  | ..                            | 113                     |
| Salmonella Infection .. ..                 | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Scarlet Fever .. ..                        | 3(3)             | 14(7)     | 8(3)        | 4(4)             | 1                  | ..                     | ..                  | ..                            | 30                      |
| Smallpox .. ..                             | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Tetanus .. ..                              | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Trachoma .. ..                             | ..               | ..        | ..          | ..               | 33                 | ..                     | ..                  | ..                            | 33                      |
| Trichinosis .. ..                          | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Tuberculosis .. ..                         | ..               | 24(17)    | 6(1)        | 3(3)             | 9(8)               | ..                     | 1                   | ..                            | 43                      |
| Typhoid Fever .. ..                        | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Typhus (Flea-, Mite- and Tick-borne) .. .. | ..               | ..        | 2           | ..               | ..                 | ..                     | ..                  | ..                            | 2                       |
| Typhus (Louse-borne) .. ..                 | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |
| Yellow Fever .. ..                         | ..               | ..        | ..          | ..               | ..                 | ..                     | ..                  | ..                            | ..                      |

<sup>1</sup> Figures in parentheses are those for the metropolitan area.

<sup>a</sup> Figures not available.

<sup>b</sup> Figures incomplete owing to absence of returns from Tasmania.

brother, Rupert Minnett, and his sister, Mrs. Norman Oakes, both live in Cremorne. They mourn his passing, and to them his friends could perhaps quote the following translation:

Happy the man and happy he alone,  
He who can call to-day his own:  
He who, secure within, can say  
"To-morrow do thy worst, for I have liv'd today!"

HORACE, Odes III, 29.

Truly did Roy Minnett live every day of a full and rich life, and family and friends are the poorer for his passing.

## Research.

### TROPICAL RESEARCH FELLOWSHIP (MEDICAL SCIENCES).

APPLICATIONS are invited by the Council of the Royal Society for a research fellowship with special reference to ill health in the tropics. The fellowship is tenable in any university, hospital or medical school or other institution approved by the Royal Society in the British Commonwealth. The successful applicant, who need not necessarily hold a medical qualification, will be expected to spend some part of the period of tenure in the tropics.

The appointment will be for two years in the first instance, from October 1, 1956, and may be renewed annually up to a total of five years. It will be subject to the conditions governing Royal Society research appointments. The stipend will be £1250 per annum.

Applications, which should be made on forms to be obtained from the Assistant Secretary, The Royal Society, Burlington House, London, W.1, should be received as early as possible, in any case not later than April 6, 1956.

## Congresses.

### INTERNATIONAL SYMPOSIUM ON VENEREAL DISEASES AND THE TREPONEMATOSES.

THE first International Symposium on Venereal Diseases and the Treponematoses will be held at the Statler Hotel, Washington, D.C., United States of America, from May 28 to June 1, 1956. It will be sponsored by the Public Health Service of the United States Department of Health, Education and Welfare, and the World Health Organization. The symposium is open to all interested doctors, scientists and professional health workers. Anyone wishing to submit a paper for consideration by the programme committee should send an abstract of the paper as soon as possible to Dr. C. A. Smith, Medical Director, Chief, Venereal Disease Program, Division of Special Health Services, Public Health Service, Department of Health, Education and Welfare, Washington 25, D.C., United States of America. Accepted papers may be presented in person or by a proxy. All abstracts to be considered for the programme must be received no later than February 1, 1956.

## Australian Medical Board Proceedings.

### TASMANIA.

THE following have been registered, pursuant to the provisions of the Medical Act, 1918, of Tasmania, as duly qualified medical practitioners: Cannon, Alleen Forsyth, M.B., B.Ch., B.A.O., 1954 (Univ. Belfast); McLaughlin, Norman Tate, M.B., Ch.B., 1953 (Univ. Birmingham); Cram, Morris, M.B., Ch.B., 1940 (Univ. Edinburgh).

## Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Coulthurst, Keith Dudley, M.B., B.S., 1955 (Univ. Sydney), 93 Garrett Street, Maroubra Junction, New South Wales.

Basser, Leon Samuel, M.B., B.S., 1946 (Univ. Sydney), M.R.A.C.P., 1950, M.R.C.P. (London), 1954, 6 Coogee Bay Road, Randwick, New South Wales.

## Diary for the Month.

- JAN. 25.—Victorian Branch, B.M.A.: Branch Council.  
JAN. 27.—Queensland Branch, B.M.A.: Council Meeting.  
FEB. 1.—Victorian Branch, B.M.A.: Branch Meeting.  
FEB. 3.—Queensland Branch, B.M.A.: General Meeting.  
FEB. 7.—New South Wales Branch, B.M.A.: Organisation and Science Committee, 8 p.m. (with Special Groups 8.30 p.m.).  
FEB. 10.—Queensland Branch, B.M.A.: Council Meeting.  
FEB. 10.—Tasmanian Branch, B.M.A.: Council Meeting.  
FEB. 13.—Victorian Branch, B.M.A.: Finance Subcommittee.  
FEB. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
FEB. 16.—Victorian Branch, B.M.A.: Executive of Branch Council.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 8 King's Park, West Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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